WEST Search History

DATE: Tuesday, June 25, 2002

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=OR			
L4	YKL adj 39	2	L4
L3	YKL39	1	L3
L2	L1 and YKL$4	1	L2
L1	(verheijden)[IN] OR (boots)[IN]	380	L1

END OF SEARCH HISTORY

(FILE 'HOME' ENTERED AT 09:46:22 ON 25 JUN 2002)

FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 09:46:36 ON 25 JUN 2002

L1 0 S FTLASAETT OR HSFTLASAETTVG
L2 211 S YKL
L3 69 S L2 AND CHONDROCYTE?
L4 42 S L3 AND (AUTOIMMUN? OR RA OR ARTHRITIS)
L5 21 DUP REM L4 (21 DUPLICATES REMOVED)
L6 19 S YKL (1N) 39
L7 10 DUP REM L6 (9 DUPLICATES REMOVED)

WO 2001 029081

WO 2000 005254

WO 2000 004917

WO [illegible]

97 400684

97 00790

Imm '99 9(4) 563-

J [illegible]

5 [illegible] 49

5736507

J Biol Chem '96 271 (32) 19[illegible]5-20

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ile medline caplus embase biosis
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

FILE 'MEDLINE' ENTERED AT 22:43:34 ON 24 JUN 2002

FILE 'CAPLUS' ENTERED AT 22:43:34 ON 24 JUN 2002
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FILE 'BIOSIS' ENTERED AT 22:43:34 ON 24 JUN 2002
COPYRIGHT (C) 2002 BIOLOGICAL ABSTRACTS INC.(R)

=> s verheijden G?/au or Boots A?/au
L1         121 VERHEIJDEN G?/AU OR BOOTS A?/AU

=> s l1 and peptide?
L2          39 L1 AND PEPTIDE?

=> s l2 and autoimmun?
L3          14 L2 AND AUTOIMMUN?

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4           10 DUP REM L3 (4 DUPLICATES REMOVED)

=> dis l4 1-10 ibib abs kwic
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Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:ssspta1644axd

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * * * * * * Welcome to STN International * * * * * * * * * *

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 NEWS  1          Web Page URLs for STN Seminar Schedule - N. America
 NEWS  2  Jan 25  BLAST(R) searching in REGISTRY available in STN on the Web
 NEWS  3  Jan 29  FSTA has been reloaded and moves to weekly updates
 NEWS  4  Feb 01  DKILIT now produced by FIZ Karlsruhe and has a new update
                  frequency
 NEWS  5  Feb 19  Access via Tymnet and SprintNet Eliminated Effective 3/31/02
 NEWS  6  Mar 08  Gene Names now available in BIOSIS
 NEWS  7  Mar 22  TOXLIT no longer available
 NEWS  8  Mar 22  TRCTHERMO no longer available
 NEWS  9  Mar 28  US Provisional Priorities searched with P in CA/CAplus
                  and USPATFULL
 NEWS 10  Mar 28  LIPINSKI/CALC added for property searching in REGISTRY
 NEWS 11  Apr 02  PAPERCHEM no longer available on STN.  Use PAPERCHEM2 instead.
 NEWS 12  Apr 08  "Ask CAS" for self-help around the clock
 NEWS 13  Apr 09  BEILSTEIN: Reload and Implementation of a New Subject Area
 NEWS 14  Apr 09  ZDB will be removed from STN
 NEWS 15  Apr 19  US Patent Applications available in IFICDB, IFIPAT, and IFIUDB
 NEWS 16  Apr 22  Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
 NEWS 17  Apr 22  BIOSIS Gene Names now available in TOXCENTER
 NEWS 18  Apr 22  Federal Research in Progress (FEDRIP) now available
 NEWS 19  Jun 03  New e-mail delivery for search results now available
 NEWS 20  Jun 10  MEDLINE Reload
 NEWS 21  Jun 10  PCTFULL has been reloaded

 NEWS EXPRESS  February 1 CURRENT WINDOWS VERSION IS V6.0d,
               CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP),
               AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
 NEWS INTER    General Internet Information
 NEWS LOGIN    Welcome Banner and News Items
 NEWS PHONE    Direct Dial and Telecommunication Network Access to STN
 NEWS WWW      CAS World Wide Web Site (general information)
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Enter NEWS followed by the item number or name to see news on that specific topic.

* * * * * * * * * * * * * * *STN Columbus* * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 22:43:19 ON 24 JUN 2002

=> file medline caplus embase biosis

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 22:43:34 ON 24 JUN 2002

FILE 'CAPLUS' ENTERED AT 22:43:34 ON 24 JUN 2002
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

FILE 'EMBASE' ENTERED AT 22:43:34 ON 24 JUN 2002

FILE 'BIOSIS' ENTERED AT 22:43:34 ON 24 JUN 2002

=> s verheijden G?/au or Boots A?/au
L1 121 VERHEIJDEN G?/AU OR BOOTS A?/AU

=> s l1 and peptide?
L2 39 L1 AND PEPTIDE?

=> s l2 and autoimmun?
L3 14 L2 AND AUTOIMMUN?

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4 10 DUP REM L3 (4 DUPLICATES REMOVED)

=> dis l4 1-10 ibib abs kwic

L4 ANSWER 1 OF 10 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:300756 CAPLUS
DOCUMENT NUMBER: 134:320857
TITLE: Modified **peptides** and peptidomimetics for use in immunotherapy
INVENTOR(S): Van Staveren, Catherina Joanna; Timmers, Cornelis Marius; Van Galen, Philippus Johannes Marie; Knegtel, Rnaldus Marcellus Alphonsus; **Boots, Anna Maria Helena**; Miltenburg, Andreas Martinus Maria
PATENT ASSIGNEE(S): *Akzo Nobel N.V., Neth.*
SOURCE: PCT Int. Appl., 52 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001029081	A1	20010426	WO 2000-EP10230	20001012

W: AE, AG, AL, AU, BA, BB, BG, BR, BZ, CA, CN, CR, CU, CZ, DM, DZ, EE, GD, GE, HR, HU, ID, IL, IN, IS, JP, KP, KR, LC, LK, LR, LT, LV, MA, MG, MK, MN, MX, MZ, NO, NZ, PL, RO, RU, SG, SI, SK, SL, TR, TT, UA, US, UZ, VN, YU, ZA, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: EP 1999-203427 A 19991018

OTHER SOURCE(S): MARPAT 134:320857

AB The invention relates to a modified **peptide** derived from formula I **peptide** H-Arg-Ser-Phe-Thr-Leu-Ala-Ser-Ser-Glu-Thr-Gly-Val-Gly-OH (**peptide** (263-275) of cartilage-derived protein human cartilage gp-39 (HC gp-39)) having general formula (II): Q-A1-A2-A3-A4-A5-A6-A7-A8-A9-A10-A11-A12-A13-Z. In general formula (II), A1 through A13 correspond with the amino acids of formula (I), Q corresponds with H and Z corresponds with OH. The modifications according to the present invention are selected from one or more of the groups a, b or c, consisting of (a) substitution of 1-6, preferably 1-4 amino acids at A1 through A13 with non-natural amino acids or .beta. amino acids; (b) substitution of one or more amide bonds with reduced amide bonds or ethylene isosteres; (c) substitutions at Q and/or Z and, optionally, (d) substitution of natural amino acids up to a total of 6 modifications. The **peptides** can be used for inducing tolerance induction in patients suffering from **autoimmune** diseases. The most potent compds. were Ac-Arg-Ser-Phe-Thr-Leu-Ala-Ser-Ser-Glu-Thr-Gly-Val-Gly-OH, Ac-Arg-Ser-Phe-Thr-Leu-Ala-Ser-Ser-Glu-Thr-Gly-Val-.psi.[CH2NH]-Gly-NH2, Ac-Arg-NhSer-Phe-Thr-Leu-Ala-Ser-Ser-Glu-Thr-Gly-Val-Gly-NH2 and Ac-Arg-NhSer-Phe-Thr-Leu-Ala-Ser-Ser-Glu-Thr-Gly-Val-.psi.[CH2NH]-Gly-NH2.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Modified **peptides** and peptidomimetics for use in immunotherapy

IN Van Staveren, Catherina Joanna; Timmers, Cornelis Marius; Van Galen, Philippus Johannes Marie; Knegtel, Rnaldus Marcellus Alphonsus; **Boots, Anna Maria Helena**; Miltenburg, Andreas Martinus Maria

AB The invention relates to a modified **peptide** derived from formula I **peptide** H-Arg-Ser-Phe-Thr-Leu-Ala-Ser-Ser-Glu-Thr-Gly-Val-Gly-OH (**peptide** (263-275) of cartilage-derived protein human cartilage gp-39 (HC gp-39)) having general formula (II): Q-A1-A2-A3-A4-A5-A6-A7-A8-A9-A10-A11-A12-A13-Z. In general formula (II), A1 through A13 correspond with the amino acids of formula (I), Q corresponds with H and Z corresponds with OH. The modifications according to the present invention are selected from one or more of the groups a, b or c, consisting of (a) substitution of 1-6, preferably 1-4 amino acids at A1 through A13 with non-natural amino acids or .beta. amino acids; (b) substitution of one or more amide bonds with reduced amide bonds or ethylene isosteres; (c) substitutions at Q and/or Z and, optionally, (d) substitution of natural amino acids up to a total of 6 modifications. The **peptides** can be used for inducing tolerance induction in patients suffering from **autoimmune** diseases. The most potent compds. were Ac-Arg-Ser-Phe-Thr-Leu-Ala-Ser-Ser-Glu-Thr-Gly-Val-Gly-OH, Ac-Arg-Ser-Phe-Thr-Leu-Ala-Ser-Ser-Glu-Thr-Gly-Val-.psi.[CH2NH]-Gly-NH2, Ac-Arg-NhSer-Phe-Thr-Leu-Ala-Ser-Ser-Glu-Thr-Gly-Val-Gly-NH2 and Ac-Arg-NhSer-Phe-Thr-Leu-Ala-Ser-Ser-Glu-Thr-Gly-Val-.psi.[CH2NH]-Gly-NH2.

ST **peptide** peptidomimetic immunotherapy; human cartilage glycoprotein 39 **peptide** immune tolerance; **autoimmune** disease immune tolerance induction **peptide**

IT Antigens
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(autoantigens, induction of specific T-cell tolerance to; modified **peptides** and peptidomimetics based on **peptide** from human cartilage glycoprotein 39 for use in immunotherapy)

IT Analysis
(biochem., diagnostic compn. contg. **peptide** and detection agent for; modified **peptides** and peptidomimetics based on **peptide** from human cartilage glycoprotein 39 for use in immunotherapy)

IT Allergy
(delayed hypersensitivity; modified **peptides** and peptidomimetics based on **peptide** from human cartilage glycoprotein 39 for use in immunotherapy)

IT Rheumatoid arthritis
(induction of specific T-cell tolerance to autoantigen of; modified **peptides** and peptidomimetics based on **peptide** from human cartilage glycoprotein 39 for use in immunotherapy)

IT **Autoimmune** disease
Diagnosis
Drug delivery systems
Immune tolerance
Immunotherapy
Peptidomimetics
(modified **peptides** and peptidomimetics based on **peptide** from human cartilage glycoprotein 39 for use in immunotherapy)

IT **Peptides**, biological studies
RL: ARG (Analytical reagent use); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PROC (Process); USES (Uses)
(modified **peptides** and peptidomimetics based on **peptide** from human cartilage glycoprotein 39 for use in immunotherapy)

IT T cell (lymphocyte)
(tolerance to autoantigen; modified **peptides** and peptidomimetics based on **peptide** from human cartilage glycoprotein 39 for use in immunotherapy)

IT 335598-47-5P 335598-48-6P 335598-49-7P 335598-50-0P 335598-51-1P 335598-52-2P 335598-54-4P 335598-55-5P 335598-56-6P 335598-57-7P 335598-58-8P 335598-60-2P
RL: ARG (Analytical reagent use); BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); SPN (Synthetic preparation); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); PROC (Process); USES (Uses)
(modified **peptides** and peptidomimetics based on **peptide** from human cartilage glycoprotein 39 for use in immunotherapy)

IT 335598-53-3 335598-59-9
RL: ARG (Analytical reagent use); BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study,

unclassified); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PROC (Process); USES (Uses)
(modified **peptides** and peptidomimetics based on **peptide** from human cartilage glycoprotein 39 for use in immunotherapy)

IT 335598-46-4P
RL: ARG (Analytical reagent use); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); RCT (Reactant); SPN (Synthetic preparation); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); PROC (Process); RACT (Reactant or reagent); USES (Uses)
(modified **peptides** and peptidomimetics based on **peptide** from human cartilage glycoprotein 39 for use in immunotherapy)

IT 335598-72-6 335598-73-7 335598-74-8
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(modified **peptides** and peptidomimetics based on **peptide** from human cartilage glycoprotein 39 for use in immunotherapy)

IT 335598-69-1 335598-70-4 335598-71-5
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(modified **peptides** and peptidomimetics based on **peptide** from human cartilage glycoprotein 39 for use in immunotherapy)

IT 335598-61-3D, conjugates with PAC-PEG-PS resin
RL: PRP (Properties); RCT (Reactant); RACT (Reactant or reagent)
(modified **peptides** and peptidomimetics based on **peptide** from human cartilage glycoprotein 39 for use in immunotherapy)

IT 335598-62-4DP, conjugates with PAL-PEG-PS resin 335598-63-5P 335598-64-6P 335598-65-7P 335598-66-8DP, conjugates with PAL-PEG-PS resin 335598-68-0DP, conjugates with PAL-PEG-PS resin
RL: PRP (Properties); RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(modified **peptides** and peptidomimetics based on **peptide** from human cartilage glycoprotein 39 for use in immunotherapy)

IT 75-65-0, reactions 298-12-4, Glyoxylic acid 2462-31-9 24424-99-5 77987-49-6 88574-06-5 97807-17-5 156939-64-9
RL: RCT (Reactant); RACT (Reactant or reagent)
(modified **peptides** and peptidomimetics based on **peptide** from human cartilage glycoprotein 39 for use in immunotherapy)

IT 141743-30-8P 258332-54-6P 335598-67-9P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(modified **peptides** and peptidomimetics based on **peptide** from human cartilage glycoprotein 39 for use in immunotherapy)

IT 199275-21-3P
RL: SPN (Synthetic preparation); PREP (Preparation)
(modified **peptides** and peptidomimetics based on **peptide** from human cartilage glycoprotein 39 for use in immunotherapy)

L4 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:84838 CAPLUS
DOCUMENT NUMBER: 132:121464
TITLE: Novel **peptides** for use in immunotherapy of **autoimmune** diseases
INVENTOR(S): **Verheijden, Gijsbertus Franciscus Maria; Boots, Anna Maria Helena**
PATENT ASSIGNEE(S): Akzo Nobel N.V., Neth.
SOURCE: PCT Int. Appl., 26 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000005254	A2	20000203	WO 1999-EP5050	19990716
WO 2000005254	A3	20000615		
W: AL, AU, BA, BB, BG, BR, CA, CN, CU, CZ, EE, GE, HU, ID, IL, IN, IS, JP, KP, KR, LC, LK, LR, LT, LV, MG, MK, MN, MX, NO, NZ, PL, RO, RU, SG, SI, SK, SL, TR, TT, UA, US, UZ, VN, YU, ZA, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9954112	A1	20000214	AU 1999-54112	19990716
BR 9912378	A	20010417	BR 1999-12378	19990716
EP 1100823	A2	20010523	EP 1999-940012	19990716
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
NO 2001000355	A	20010320	NO 2001-355	20010122

PRIORITY APPLN. INFO.: EP 1998-202470 A 19980723
WO 1999-EP5050 W 19990716

AB The invention relates to the use of novel **peptides** in a **peptide** induced tolerance therapy to prevent **autoimmune** disorders and in particular their use in treatment of chronic destruction of articular cartilage. The invention furthermore embraces pharmaceutical compns. comprising said **peptides** and a diagnostic method for the detection of autoreactive T cells in a test sample.

TI Novel **peptides** for use in immunotherapy of **autoimmune** diseases

IN **Verheijden, Gijsbertus Franciscus Maria; Boots, Anna Maria Helena**

AB The invention relates to the use of novel **peptides** in a **peptide** induced tolerance therapy to prevent **autoimmune** disorders and in particular their use in treatment of chronic destruction of articular cartilage. The invention furthermore embraces pharmaceutical compns. comprising said **peptides** and a diagnostic method for the detection of autoreactive T cells in a test sample.

ST **autoimmune** articular cartilage destruction tolerance immunotherapy; autoantigen tolerance **autoimmune** articular cartilage destruction

IT Histocompatibility antigens
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL

(Biological study); PROC (Process)
(HLA-DR, DRB1*0401; autoantigenic **peptides** for inducing tolerance for immunotherapy of **autoimmune** diseases)

IT Cartilage
(articular, chronic destruction; autoantigenic **peptides** for inducing tolerance for immunotherapy of **autoimmune** diseases)

IT **Autoimmune** disease
Immunotherapy
Protein sequences
Rheumatoid arthritis
(autoantigenic **peptides** for inducing tolerance for immunotherapy of **autoimmune** diseases)

IT Antigens
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(autoantigens; autoantigenic **peptides** for inducing tolerance for immunotherapy of **autoimmune** diseases)

IT Drug delivery systems
(carriers; autoantigenic **peptides** for inducing tolerance for immunotherapy of **autoimmune** diseases)

IT Musculoskeletal diseases
(cartilage, articular; autoantigenic **peptides** for inducing tolerance for immunotherapy of **autoimmune** diseases)

IT Cartilage
(disease, articular; autoantigenic **peptides** for inducing tolerance for immunotherapy of **autoimmune** diseases)

IT Glycoproteins, specific or class
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(gp39, human chondrocyte protein; autoantigenic **peptides** for inducing tolerance for immunotherapy of **autoimmune** diseases)

IT Immune tolerance
(specific T cell; autoantigenic **peptides** for inducing tolerance for immunotherapy of **autoimmune** diseases)

IT T cell (lymphocyte)
(specific tolerance; autoantigenic **peptides** for inducing tolerance for immunotherapy of **autoimmune** diseases)

IT 9001-06-3, Chitotriosidase
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(autoantigenic **peptides** for inducing tolerance for immunotherapy of **autoimmune** diseases)

IT 178274-47-0 256450-45-0 256450-46-1 256450-47-2
RL: PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(autoantigenic **peptides** for inducing tolerance for immunotherapy of **autoimmune** diseases)

IT 148740-87-8
RL: PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(pos. control; autoantigenic **peptides** for inducing tolerance for immunotherapy of **autoimmune** diseases)

L4 ANSWER 3 OF 10 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:84638 CAPLUS
DOCUMENT NUMBER: 132:121462
TITLE: Use of human cartilage (HC) gp-39 in immune diseases
INVENTOR(S): Miltenburg, Andreas Martinus Maria; **Boots, Anna Maria Helena**
PATENT ASSIGNEE(S): Akzo Nobel N.V., Neth.
SOURCE: PCT Int. Appl., 29 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000004917	A2	20000203	WO 1999-EP5331	19990719
WO 2000004917	A3	20000720		
W: AL, AU, BA, BB, BG, BR, CA, CN, CU, CZ, EE, GE, HU, ID, IL, IN, IS, JP, KP, KR, LC, LK, LR, LT, LV, MG, MK, MN, MX, NO, NZ, PL, RO, RU, SG, SI, SK, SL, TR, TT, UA, US, UZ, VN, YU, ZA, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9952875	A1	20000214	AU 1999-52875	19990719
EP 1100526	A2	20010523	EP 1999-938340	19990719
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

PRIORITY APPLN. INFO.: EP 1998-202471 A 19980723
WO 1999-EP5331 W 19990719

AB The present invention relates to the use of HC gp-39 to prevent (auto)immune disease or inflammatory diseases, e.g. rheumatoid arthritis. More specifically, HC gp-39 or fragments thereof can be used to modulate the reactivity of lymphocytes which are reactive to antigens other than HC gp-39 but which are present in the same tissue as where HC gp-39 is being expressed.

IN Miltenburg, Andreas Martinus Maria; **Boots, Anna Maria Helena**

ST human cartilage gp39 **autoimmune** inflammatory disease; rheumatoid arthritis gp39 **peptide**

IT Glycoproteins, specific or class
RL: PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(CMP (cartilage matrix protein), gp-39; human cartilage gp-39 **peptides** for preventing (auto)immune and inflammatory diseases)

IT Immunity
(disorder; human cartilage gp-39 **peptides** for preventing (auto)immune and inflammatory diseases)

IT Glycoproteins, specific or class
RL: PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(gp39; human cartilage gp-39 **peptides** for preventing (auto)immune and inflammatory diseases)

IT **Autoimmune** disease
Inflammation
Protein sequences
Rheumatoid arthritis
(human cartilage gp-39 **peptides** for preventing (auto)immune and inflammatory diseases)

IT Lymphocyte

(modulation; human cartilage gp-39 **peptides** for preventing (auto)immune and inflammatory diseases)

IT 178274-42-5 178274-43-6 178274-44-7 178274-45-8 178274-46-9 178274-47-0 178274-48-1 178274-49-2
RL: PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(human cartilage gp-39 **peptides** for preventing (auto)immune and inflammatory diseases)

L4 ANSWER 4 OF 10 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 1999250342 MEDLINE
DOCUMENT NUMBER: 99250342 PubMed ID: 10233745
TITLE: T-cell anergy induced by clonotype-specific antibodies: modulation of an autoreactive human T-cell clone in vitro.
AUTHOR: Steenbakkers P G; **Boots A M**; Rijnders A W
CORPORATE SOURCE: Department of Immunology, N. V. Organon, Oss, The Netherlands.
SOURCE: IMMUNOLOGY, (1999 Apr) 96 (4) 586-94.
Journal code: 0374672. ISSN: 0019-2805.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 19990816
Last Updated on STN: 19990816
Entered Medline: 19990730

AB Monoclonal antibodies (mAb) specific for the clonotype of an autoreactive T cell may be useful reagents in the modulation of **autoimmune** disease. We have previously reported the generation of a set of mAb specific for the clonotypic structure of a human T-cell clone recognizing an epitope of human cartilage gp-39. This glycoprotein was recently identified as a candidate autoantigen in rheumatoid arthritis. Here, we demonstrate for the first time that small amounts of immobilized anticlonotype mAb can induce anergy in the autoreactive clone. Following the anergic stimulus, T cells failed to proliferate upon restimulation as a result of a lack of interleukin-2 (IL-2) gene transcription. In addition, a diminished interferon-gamma (IFN-gamma) production was found. Our data indicate that anergy was not a result of T-cell receptor (TCR) downmodulation or the absence of free TCR. The anergic state was induced independent of costimulation or the presence of IL-2 and no protein synthesis was required for the induction of anergy. Anticlonotype mAb-induced anergy was prevented by cyclosporin A, suggesting that active signalling via the calcium/calcineurin pathway was required for the induction of anergy. In coculture experiments, anergic T cells were found to suppress the response of reactive cells from the same clone. This bystander suppression led to 90% inhibition of **peptide**-induced proliferation. Together, these findings suggest that mAb to the clonotypic structure of autoreactive T cells may be suitable reagents for the functional inactivation of these T cells in **autoimmune** diseases.

AU Steenbakkers P G; **Boots A M**; Rijnders A W

AB Monoclonal antibodies (mAb) specific for the clonotype of an autoreactive T cell may be useful reagents in the modulation of **autoimmune** disease. We have previously reported the generation of a set of mAb specific for the clonotypic structure of a human. . . found to suppress the response of reactive cells from the same clone. This bystander suppression led to 90% inhibition of **peptide**-induced proliferation. Together, these findings suggest that mAb to the clonotypic structure of autoreactive T cells may be suitable reagents for the functional inactivation of these T cells in **autoimmune** diseases.

L4 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 2
ACCESSION NUMBER: 1998:785564 CAPLUS
DOCUMENT NUMBER: 130:37290
TITLE: Proteins and novel **peptides** derived from autoantigen for use in immunotherapy of **autoimmune** diseases
INVENTOR(S): **Boots, Anna Maria Helena**; **Verheijden, Gijsbertus Franciscus Maria**; Bos, Ebo Sybren
PATENT ASSIGNEE(S): Akzo Nobel N.V., Neth.
SOURCE: U.S., 19 pp., Cont.-in-part of U.S. 5,736,507.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5843449	A	19981201	US 1996-634493	19960418
US 5736507	A	19980407	US 1996-619645	19960325
CA 2251584	AA	19971030	CA 1997-2251584	19970415
WO 9740149	A1	19971030	WO 1997-EP1903	19970415
W: AM, AU, BB, BG, BR, CA, CN, CZ, EE, GE, HU, IS, JP, KG, KP, KR, LK, LR, LT, LV, MD, MG, MN, MX, NO, NZ, PL, RO, RU, SG, SI, SK, TR, TT, UA, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9723869	A1	19971112	AU 1997-23869	19970415
AU 724547	B2	20000928		
EP 904369	A1	19990331	EP 1997-919370	19970415
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
CN 1216582	A	19990512	CN 1997-193881	19970415
BR 9708714	A	19990803	BR 1997-8714	19970415
JP 2000509265	T2	20000725	JP 1997-537477	19970415
KR 2000005476	A	20000125	KR 1998-8253	19981015
NO 9804835	A	19981216	NO 1998-4835	19981016

PRIORITY APPLN. INFO.:
US 1996-619645 A2 19960325
EP 1994-203128 A 19941027
EP 1995-200886 A 19950407
WO 1995-EP4201 W 19951025
US 1996-634493 A 19960418
WO 1997-EP1903 W 19970415

AB The present invention relates to novel **peptides** derived from the autoantigen HC gp-39, said **peptides** comprising at least one of the amino acid sequences FGRSFTILAS (SEQ ID No. 1), FTLASSETG (SEQ ID No. 2), YDDQESVKS (SEQ ID No. 3) and FSKIASNTQ (SEQ ID No. 4). The **peptides** resemble MHC Class II restricted T-cell epitopes present on the autoantigen HC gp-39 in articular cartilage. HC gp-39, proteins comprising an amino acid sequence which exhibits at least 50% homol. with the amino acid sequence YKLVCYYTSWSQYREGDGSCFPDALDRFLCTHIIYSFANISND (SEQ

ID No: 10) and said **peptides** can be used in antigen-specific treatment of articular cartilage destruction in **autoimmune** diseases in mammals to induce systemic tolerance of the immune system. The autoantigen HC gp-39, proteins comprising an amino acid sequence which exhibits at least 50% homol. with the amino acid sequence YKLVCYYTSWSQYREGDGSCFPDALDRFLCTHIIYSFANISND (SEQ ID NO: 10) and said **peptides** are also suitable to induce arthritis in animals, preferably mice. The invention furthermore relates to pharmaceutical compns. comprising said autoantigen and/or said **peptides**, a diagnostic method for the detection of autoreactive T cells in a test sample and test kits to be used in said method. For treating T cell-mediated cartilage destruction disease (e.g. arthritis or rheumatoid arthritis), the T cell-specific tolerance-inducing **peptide** or protein can also be selected from the group consisting of pig heparin binding 38 kDa protein, bovine 39 kDa whey protein, murine breast regressing 39 kDa protein (brp39), human oviduct-specific glycoprotein, murine oviduct-specific glycoprotein, hamster oviduct-specific glycoprotein, bovine oviduct-specific glycoprotein, human chitotriosidase precursor and their fragments.

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Proteins and novel **peptides** derived from autoantigen for use in immunotherapy of **autoimmune** diseases

IN **Boots, Anna Maria Helena; Verheijden, Gijsbertus Franciscus Maria**; Bos, Ebo Sybren

AB The present invention relates to novel **peptides** derived from the autoantigen HC gp-39, said **peptides** comprising at least one of the amino acid sequences FGRSFTILAS (SEQ ID No. 1), FTLASSETG (SEQ ID No. 2), YDDQESVKS (SEQ ID No. 3) and FSKIASNTQ (SEQ ID No. 4). The **peptides** resemble MHC Class II restricted T-cell epitopes present on the autoantigen HC gp-39 in articular cartilage. HC gp-39, proteins comprising an amino acid sequence which exhibits at least 50% homol. with the amino acid sequence YKLVCYYTSWSQYREGDGSCFPDALDRFLCTHIIYSFANISND (SEQ ID No: 10) and said **peptides** can be used in antigen-specific treatment of articular cartilage destruction in **autoimmune** diseases in mammals to induce systemic tolerance of the immune system. The autoantigen HC gp-39, proteins comprising an amino acid sequence which exhibits at least 50% homol. with the amino acid sequence YKLVCYYTSWSQYREGDGSCFPDALDRFLCTHIIYSFANISND (SEQ ID NO: 10) and said **peptides** are also suitable to induce arthritis in animals, preferably mice. The invention furthermore relates to pharmaceutical compns. comprising said autoantigen and/or said **peptides**, a diagnostic method for the detection of autoreactive T cells in a test sample and test kits to be used in said method. For treating T cell-mediated cartilage destruction disease (e.g. arthritis or rheumatoid arthritis), the T cell-specific tolerance-inducing **peptide** or protein can also be selected from the group consisting of pig heparin binding 38 kDa protein, bovine 39 kDa whey protein, murine breast regressing 39 kDa protein (brp39), human oviduct-specific glycoprotein, murine oviduct-specific glycoprotein, hamster oviduct-specific glycoprotein, bovine oviduct-specific glycoprotein, human chitotriosidase precursor and their fragments.

ST autoantigen gp39 **autoimmune** cartilage destruction disease; rheumatoid arthritis autoantigen HC gp39 **peptide**

IT Proteins, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(38,000-mol.-wt.; **peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

IT Proteins, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(39,000-mol.-wt.; **peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

IT Histocompatibility antigens
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(MHC (major histocompatibility complex), class II; **peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

IT Epitopes
(T cell; **peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

IT Antigens
RL: ADV (Adverse effect, including toxicity); ARU (Analytical role, unclassified); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study)
(autoantigens, HC gp39; **peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

IT Proteins, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(breast regressing 39 kDa proteins; **peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

IT Cartilage
Cartilage
(degeneration; **peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

IT T cell (lymphocyte)
(epitope; **peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

IT Oviduct
(glycoprotein specific to; **peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

IT Proteins, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL

(Biological study); USES (Uses)
(heparin-binding, 38 kDa; **peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

IT Drug delivery systems
(injections; **peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

IT Drug delivery systems
(nasal, intra-; **peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

IT Drug delivery systems
(oral; **peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

IT Glycoproteins, general, biological studies
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(oviduct-specific; **peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

IT Animal
Arthritis
Autoimmune disease
Cattle
Hamster
Immune tolerance
Mammal (Mammalia)
Mouse
Protein sequences
Rheumatoid arthritis
Rodent
(**peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

IT Proteins, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(secretory, YM-1 precursor; **peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

IT Proteins, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(whey, 39 kDa; **peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

IT 198841-51-9
RL: ADV (Adverse effect, including toxicity); ARU (Analytical role, unclassified); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study)
(**peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

IT 172253-32-6
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

IT 148740-87-8 178274-42-5 178274-43-6 178274-44-7 178274-45-8
178274-46-9 178274-47-0 178274-48-1 178274-49-2
RL: PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**peptides** derived from autoantigen HC gp39 for inducing T cell-specific tolerance and for treating **autoimmune** disease such as cartilage destruction diseases and arthritis or rheumatoid arthritis)

L4 ANSWER 6 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:106274 BIOSIS
DOCUMENT NUMBER: PREV200200106274
TITLE: **Peptides** derived from autoantigen for use in immunotherapy of **autoimmune** diseases.
AUTHOR(S): **Boots, A. M. H.; Verheijden, G. F. M.**
CORPORATE SOURCE: Verlengde Torenstraat Netherlands
ASSIGNEE: AKZO NOBEL N.V.
PATENT INFORMATION: US 5736507 April 7, 1998
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (April 7, 1998) Vol. 1209, No. 1, pp. 492. ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
TI **Peptides** derived from autoantigen for use in immunotherapy of **autoimmune** diseases.
AU **Boots, A. M. H.; Verheijden, G. F. M.**
IT Sequence Data
AMINO ACID SEQUENCE
IT Miscellaneous Descriptors
CHEMICAL FORMULA; HUMAN CARTILAGE **PEPTIDE**; IMMUNOLOGIC AGENT; PHARMACEUTICALS

L4 ANSWER 7 OF 10 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:717938 CAPLUS
DOCUMENT NUMBER: 128:2901
TITLE: *Novel* ***peptides*** *suitable for use in antigen* specific immunosuppressive therapy
INVENTOR(S): **Boots, Anna Maria Helena; Verheijden, Gijsbertus Franciscus Maria**
PATENT ASSIGNEE(S): Akzo Nobel N.V., Neth.; Boots, Anna Maria Helena; Verheijden, Gijsbertus Franciscus Maria
SOURCE: PCT Int. Appl., 82 pp.

CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9740068	A1	19971030	WO 1997-EP2051	19970422
W: AU, BR, CA, CN, CZ, HU, JP, KR, MX, NO, NZ, PL, RU, SG, TR, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
ZA 9703071	A	19980805	ZA 1997-3071	19970410
CA 2251680	AA	19971030	CA 1997-2251680	19970422
AU 9727685	A1	19971112	AU 1997-27685	19970422
AU 719481	B2	20000511		
CN 1216551	A	19990512	CN 1997-194031	19970422
EP 920451	A1	19990609	EP 1997-921707	19970422
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
BR 9708744	A	19990803	BR 1997-8744	19970422
JP 2000510106	T2	20000808	JP 1997-537744	19970422
KR 2000010561	A	20000215	KR 1998-708415	19981021
NO 9804937	A	19981201	NO 1998-4937	19981023
US 6184204	B1	20010206	US 1998-171705	19981023

PRIORITY APPLN. INFO.: EP 1996-201106 A 19960424
WO 1997-EP2051 W 19970422

AB The invention relates to **peptides** consisting of 16 to 55 amino acid residues, and useful in diagnosis for detecting activated autoreactive T cells in the individual. The **peptides** are also useful in the treatment of **autoimmune** disease, e.g. T-cell *mediated destruction of articular cartilage. Administration of* pharmaceutical compns. based on these **peptides** can be used to induce systemic immunol. tolerance to the autoantigens under attack of the autoreactive T-cells.

TI Novel **peptides** suitable for use in antigen specific immunosuppressive therapy

IN **Boots, Anna Maria Helena**; **Verheijden, Gijsbertus Franciscus Maria**

AB The invention relates to **peptides** consisting of 16 to 55 amino acid residues, and useful in diagnosis for detecting activated autoreactive T cells in the individual. The **peptides** are also useful in the treatment of **autoimmune** disease, e.g. T-cell mediated destruction of articular cartilage. Administration of pharmaceutical compns. based on these **peptides** can be used to induce systemic immunol. tolerance to the autoantigens under attack of the autoreactive T-cells.

ST antigen immunosuppressant autoreactive T cell tolerance; **autoimmune** disease articular cartilage immunosuppressive **peptide**

IT Cartilage
(articular, destruction; immunosuppressive antigen **peptides** for inhibiting autoreactive T cell and for treating and diagnosing **autoimmune** disease)

IT Antigens
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(autoantigens; immunosuppressive antigen **peptides** for inhibiting autoreactive T cell and for treating and diagnosing **autoimmune** disease)

IT T cell (lymphocyte)
(autoreactive; immunosuppressive antigen **peptides** for inhibiting autoreactive T cell and for treating and diagnosing **autoimmune** disease)

IT **Autoimmune** disease
Immune tolerance
Immunosuppressants
Protein sequences
(immunosuppressive antigen **peptides** for inhibiting autoreactive T cell and for treating and diagnosing **autoimmune** *disease*)

IT Antigens
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(immunosuppressive antigen **peptides** for inhibiting autoreactive T cell and for treating and diagnosing **autoimmune** disease)

IT Mononuclear cell (leukocyte)
(peripheral blood; immunosuppressive antigen **peptides** for inhibiting autoreactive T cell and for treating and diagnosing **autoimmune** disease)

IT Blood
(*peripheral; immunosuppressive antigen* **peptides** for inhibiting autoreactive T cell and for treating and diagnosing **autoimmune** disease)

IT 198880-51-2 198880-52-3 198880-53-4 198880-54-5 198880-55-6
198880-56-7 198880-57-8 198880-58-9 198880-59-0 198880-60-3
198880-61-4 198880-62-5 198880-63-6 198880-64-7 198880-65-8
198880-67-0 198880-69-2 198880-70-5 198880-71-6 198880-72-7
198880-73-8 198880-74-9 198880-75-0 198880-76-1 198880-77-2
198880-78-3 198880-79-4 198880-80-7 198880-81-8 198880-82-9
198880-83-0 198880-84-1 198880-85-2 198880-86-3 198880-87-4
198880-88-5 198880-89-6 198880-90-9 198880-91-0 198880-92-1
198880-93-2 198880-94-3 198880-95-4 198880-96-5
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(immunosuppressive antigen **peptides** for inhibiting autoreactive T cell and for treating and diagnosing **autoimmune** disease)

L4 ANSWER 8 OF 10 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:151549 CAPLUS
DOCUMENT NUMBER: 126:152794
TITLE: **Peptides** for use in treatment of T-cell mediated cartilage destruction in **autoimmune** diseases
INVENTOR(S): **Verheijden, Gijsbertus Francisc**; **Boots, Anna Maria Helena**
PATENT ASSIGNEE(S): Akzo Nobel N.V., Neth.; Verheijden, Gijsbertus Franciscus Maria; Boots, Anna Maria Helena
SOURCE: PCT Int. Appl., 22 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: *English*
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9700270	A1	19970103	WO 1996-EP2605	19960617
W: AL, AM, AU, BB, BG, BR, CA, CN, CZ, EE, GE, HU, IS, JP, KG, KP, KR, LK, LR, LT, LV, MD, MG, MK, MN, MX, NO, NZ, PL, RO, SG, SI, SK, TR, TT, UA, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2221981	AA	19970103	CA 1996-2221981	19960617
AU 9662246	A1	19970115	AU 1996-62246	19960617
AU 704942	B2	19990506		
EP 833842	A1	19980408	EP 1996-920822	19960617
EP 833842	B1	19990929		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
CN 1188483	A	19980722	CN 1996-194869	19960617
BR 9608626	A	19990615	BR 1996-8626	19960617
JP 11507919	T2	19990713	JP 1996-502649	19960617
AT 185151	E	19991015	AT 1996-920822	19960617
ES 2139363	T3	20000201	ES 1996-920822	19960617
ZA 9605163	A	19970123	ZA 1996-5163	19960618
NO 9705968	A	19971218	NO 1997-5968	19971218
PRIORITY APPLN. INFO.:			EP 1995-201656 A	19950619
			WO 1996-EP2605 W	19960617

AB The invention relates to the use of novel **peptides** in a **peptide**-induced tolerance therapy for the induction of tolerance to autoaggressive T cells assocd. with T-cell mediated articular cartilage destruction in **autoimmune** diseases, more specifically arthritis. The invention furthermore embraces pharmaceutical compns. comprising said **peptides** and a diagnostic method for the detection of autoreactive T cells in a test sample, said T cells being assocd. with T-cell mediated articular cartilage destruction in **autoimmune** diseases and test kits to be used in said method.

TI **Peptides** for use in treatment of T-cell mediated cartilage destruction in **autoimmune** diseases

IN **Verheijden, Gijsbertus Francisc**; **Boots, Anna Maria Helena**

AB The invention relates to the use of novel **peptides** in a **peptide**-induced tolerance therapy for the induction of tolerance to autoaggressive T cells assocd. with T-cell mediated articular cartilage destruction in **autoimmune** diseases, more specifically arthritis. The invention furthermore embraces pharmaceutical compns. comprising said **peptides** and a diagnostic method for the detection of autoreactive T cells in a test sample, said T cells being assocd. with T-cell mediated articular cartilage destruction in **autoimmune** diseases and test kits to be used in said method.

ST **autoimmune** disease cartilage destruction **peptide** therapy

IT **Autoimmune** disease
Cartilage
Rheumatoid arthritis
T cell (lymphocyte)
(**peptides** for use in treatment of T-cell mediated cartilage destruction in **autoimmune** diseases)

IT 186827-57-6 186827-58-7 186827-59-8 186827-60-1
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**peptides** for use in treatment of T-cell mediated cartilage destruction in **autoimmune** diseases)

L4 ANSWER 9 OF 10 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 1998037624 EMBASE

TITLE: Selection of self-reactive **peptides** within human aggrecan by use of a HLA-DRB1*0401 **peptide** binding motif.

AUTHOR: **Boots A.M.H.**; **Verheijden G.F.M.**; Schoningh R.; Van Staveren C.J.; Bos E.; Elewaut D.; De Keyser F.; Veys E.; Joosten I.; Rijnders A.W.M.

CORPORATE SOURCE: Dr. A.M.H. Boots, Department of Immunology, RH165, NV Organon, P.O. Box 20, 5340 BH Oss, Netherlands

SOURCE: Journal of Autoimmunity, (1997) 10/6 (569-578). Refs: 40
ISSN: 0896-8411 CODEN: JOAUEP

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 026 Immunology, Serology and Transplantation

LANGUAGE: English

SUMMARY LANGUAGE: English

AB The pathogenesis of joint destruction in rheumatoid arthritis remains ill-defined. Joint destruction is thought to be the result of tissue damage mediated by T cells. The mere presence of articular cartilage appears responsible for sustaining chronic synovitis and thereby forwards a role for cartilage-responsive T cells in RA. Taking advantage of the positive DRB1*0401 association with RA susceptibility, we reasoned that T-cell recognition of autoantigens in RA would be restricted by DRB1*0401-encoded molecules. A DR4 (B1*0401) **peptide** binding motif was used for the identification of putative T-cell epitopes within human aggrecan, a candidate autoantigen. Thirteen **peptides** were synthesized and tested for binding DRB1*0401 or 0404-encoded molecules. Selected binders were tested for induction of proliferative responses in peripheral blood mononuclear cells from donors carrying the DR4 or DR1 specificity. Both healthy and RA donors responded to human aggrecan-derived **peptides**, thereby identifying these sequences as T-cell epitopes. Interestingly, responses to aggrecan-derived epitopes were significantly decreased in RA patients compared to controls. This was not due to an overall hyporesponsiveness of RA patients since responses to a recall antigen or mitogen did not differ from controls. The data suggest that in RA, aggrecan-specific T cells may exist in a different stage of activation or may have left the periphery to home to the joint.

TI Selection of self-reactive **peptides** within human aggrecan by use of a HLA-DRB1*0401 **peptide** binding motif.

AU **Boots A.M.H.**; **Verheijden G.F.M.**; Schoningh R.; Van Staveren C.J.; Bos E.; Elewaut D.; De Keyser F.; Veys E.; Joosten I.; Rijnders A.W.M.

AB . . . RA susceptibility, we reasoned that T-cell recognition of autoantigens in RA would be restricted by DRB1*0401-encoded molecules. A DR4 (B1*0401) **peptide** binding motif was used for the identification of putative T-cell epitopes within human aggrecan, a candidate autoantigen. Thirteen **peptides** were synthesized and tested for binding DRB1*0401 or 0404-encoded molecules. Selected binders were tested for induction of proliferative responses in. . . blood

mononuclear cells from donors carrying the DR4 or DR1 specificity. Both healthy and RA donors responded to human aggrecan-derived **peptides**, thereby identifying these sequences as T-cell epitopes. Interestingly, responses to aggrecan-derived epitopes were significantly decreased in RA patients compared to. . .

CT Medical Descriptors:
***peptide analysis**
autoimmunity
t lymphocyte activation
pathogenesis
tissue injury
molecular recognition
mononuclear cell
antigen specificity
antigen presentation
cell proliferation
human
human cell
article
priority journal
*aggrecan
*HLA DR antigen

L4 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:397369 CAPLUS
DOCUMENT NUMBER: 125:49310
TITLE: Novel **peptides** derived from the articular cartilage autoantigen HC gp-39 for use in immunotherapy of **autoimmune** diseases
INVENTOR(S): **Boots, Anna Maria Helena; Verheijden, Gijsbertus Franciscus Maria**
PATENT ASSIGNEE(S): Akzo Nobel N.V., Neth.
SOURCE: PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9613517	A1	19960509	WO 1995-EP4201	19951025
W: AU, BR, CA, CN, FI, HU, JP, KR, MX, NO, NZ, PL, RU, US				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
IL 115744	A1	20000716	IL 1995-115744	19951024
AU 9539252	A1	19960523	AU 1995-39252	19951025
AU 696827	B2	19980917		
EP 733065	A1	19960925	EP 1995-937008	19951025
EP 733065	B1	19990317		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
HU 74847	A2	19970228	HU 1996-1401	19951025
HU 218027	B	20000528		
JP 09507861	T2	19970812	JP 1995-514306	19951025
BR 9506377	A	19970916	BR 1995-6377	19951025
CN 1168677	A	19971224	CN 1995-190944	19951025
AT 177756	E	19990415	AT 1995-937008	19951025
ES 2130672	T3	19990701	ES 1995-937008	19951025
ZA 9509123	A	19970123	ZA 1995-9123	19951027
US 5736507	A	19980407	US 1996-619645	19960325
FI 9602619	A	19960625	FI 1996-2619	19960625
NO 9602695	A	19960626	NO 1996-2695	19960626

PRIORITY APPLN. INFO.: EP 1994-203128 A 19941027
EP 1995-200886 A 19950407
WO 1995-EP4201 W 19951025

AB Novel **peptides** derived from the autoantigen HC gp-39 including at least one of the fragments FGRSFTLAS, FTLASSETC, YDDQESVKS or FSKIASNTQ are described for use in the induction of immune tolerance in the treatment of **autoimmune** disease. The **peptides** resemble MHC Class II restricted T-cell epitopes present on the autoantigen HC gp-39 in articular cartilage. HC gp-39 and these **peptides** can be used in antigen-specific treatment of articular cartilage destruction in **autoimmune** diseases to induce tolerance of the immune system. The autoantigen HC gp-39 and these **peptides** are also suitable to induce arthritis in non-human animals, preferably mice. The invention furthermore relates to pharmaceutical compns. comprising said autoantigen and/or said **peptides**, a diagnostic method for the detection of autoreactive T cells in a test sample and test kits to be used in said method. The use of these **peptides** to induce immune tolerance in mice is demonstrated.

TI Novel **peptides** derived from the articular cartilage autoantigen HC gp-39 for use in immunotherapy of **autoimmune** diseases

IN **Boots, Anna Maria Helena; Verheijden, Gijsbertus Franciscus Maria**

AB Novel **peptides** derived from the autoantigen HC gp-39 including at least one of the fragments FGRSFTLAS, FTLASSETC, YDDQESVKS or FSKIASNTQ are described for use in the induction of immune tolerance in the treatment of **autoimmune** disease. The **peptides** resemble MHC Class II restricted T-cell epitopes present on the autoantigen HC gp-39 in articular cartilage. HC gp-39 and these **peptides** can be used in antigen-specific treatment of articular cartilage destruction in **autoimmune** diseases to induce tolerance of the immune system. The autoantigen HC gp-39 and these **peptides** are also suitable to induce arthritis in non-human animals, preferably mice. The invention furthermore relates to pharmaceutical compns. comprising said autoantigen and/or said **peptides**, a diagnostic method for the detection of autoreactive T cells in a test sample and test kits to be used in said method. The use of these **peptides** to induce immune tolerance in mice is demonstrated.

ST gp39 **peptide** autoantigen **autoimmune** disease therapy

IT Immune tolerance
(induction of; novel **peptides** derived from articular cartilage autoantigen HC gp-39 for use in immunotherapy of **autoimmune** diseases)

IT Lymphocyte
(T-cell, autoreactive, detection of; novel **peptides** derived from articular cartilage autoantigen HC gp-39 for use in immunotherapy of **autoimmune** diseases)

IT Inflammation inhibitors
(antiarthritics, novel **peptides** derived from articular cartilage autoantigen HC gp-39 for use in immunotherapy of **autoimmune** diseases)

IT Cartilage
(articular, preventing destruction in **autoimmune** disease of;

novel **peptides** derived from articular cartilage autoantigen HC gp-39 for use in immunotherapy of **autoimmune** diseases)

IT Antigens
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(auto-, cartilage HC gp-39; novel **peptides** derived from articular cartilage autoantigen HC gp-39 for use in immunotherapy of **autoimmune** diseases)

IT *Glycoproteins, specific or class*
RL: PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(gp39, cartilage autoantigen; novel **peptides** derived from articular cartilage autoantigen HC gp-39 for use in immunotherapy of **autoimmune** diseases)

IT Arthritis
(rheumatoid, novel **peptides** derived from articular cartilage autoantigen HC gp-39 for use in immunotherapy of **autoimmune** diseases)

IT 178274-42-5D, analogs, derivs. 178274-43-6D, analogs, derivs.
178274-44-7D, analogs, derivs. 178274-45-8D, analogs, derivs.
178274-46-9D, analogs, derivs. 178274-47-0D, analogs, derivs.
178274-48-1D, analogs, derivs. 178274-49-2D, analogs, derivs.
RL: PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(novel **peptides** derived from articular cartilage autoantigen HC gp-39 for use in immunotherapy of **autoimmune** diseases)

=> end
ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
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COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	31.95	32.16
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
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NEWS 3	Jan 29	FSTA has been reloaded and moves to weekly updates
NEWS 4	Feb 01	DKILIT now produced by FIZ Karlsruhe and has a new update frequency
NEWS 5	Feb 19	Access via Tymnet and SprintNet Eliminated Effective 3/31/02
NEWS 6	Mar 08	Gene Names now available in BIOSIS
NEWS 7	Mar 22	TOXLIT no longer available
NEWS 8	Mar 22	TRCTHERMO no longer available
NEWS 9	Mar 28	US Provisional Priorities searched with P in CA/CAplus and USPATFULL
NEWS 10	Mar 28	LIPINSKI/CALC added for property searching in REGISTRY
NEWS 11	Apr 02	PAPERCHEM no longer available on STN. Use PAPERCHEM2 instead.
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NEWS 16	Apr 22	Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
NEWS 17	Apr 22	BIOSIS Gene Names now available in TOXCENTER
NEWS 18	Apr 22	Federal Research in Progress (FEDRIP) now available
NEWS 19	Jun 03	New e-mail delivery for search results now available
NEWS 20	Jun 10	MEDLINE Reload
NEWS 21	Jun 10	PCTFULL has been reloaded
NEWS EXPRESS		February 1 CURRENT WINDOWS VERSION IS V6.0d, CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP), AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
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	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

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FILE 'CAPLUS' ENTERED AT 09:46:36 ON 25 JUN 2002

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=> s FTLASAETT or HSFTLASAETTVG
L1 0 FTLASAETT OR HSFTLASAETTVG

=> s ykl
L2 211 YKL

=> s l2 and chondrocyte?
L3 69 L2 AND CHONDROCYTE?

=> s l3 and (autoimmun? or RA or arthritis)
L4 42 L3 AND (AUTOIMMUN? OR RA OR ARTHRITIS)

=> duup rem l4
DUUP IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5 21 DUP REM L4 (21 DUPLICATES REMOVED)

=> dis l5 1-21 ibib abs

L5 ANSWER 1 OF 21 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001147218 EMBASE
TITLE: Regulation of **YKL**-40 production by human articular **chondrocytes**.
AUTHOR: Johansen J.S.; Olee T.; Price P.A.; Hashimoto S.; Ochs R.L.; Lotz M.
CORPORATE SOURCE: Dr. J.S. Johansen, Department of Internal Medicine, Division of Rheumatology 232, Hvidovre Hospital, Kettegard alle 30, DK-2650 Hvidovre, Denmark
SOURCE: Arthritis and Rheumatism, (2001) 44/4 (826-837).
Refs: 40
ISSN: 0004-3591 CODEN: ARHEAW
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
029 Clinical Biochemistry
031 Arthritis and Rheumatism
033 Orthopedic Surgery
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Objective. **YKL**-40 (human cartilage glycoprotein 39) is one of the most abundant proteins secreted by cultured **chondrocytes**. The objectives of the present study were to identify regulators of **YKL**-40 production in cartilage and **chondrocytes** and to map the localization of **YKL**-40 in **chondrocytes**. Methods. Human articular **chondrocytes** and cartilage explants (obtained from subjects at autopsy, from a tissue bank, and from osteoarthritis [OA] patients undergoing total joint replacement surgery) were stimulated with cytokines, growth factors, and other agents. **YKL**-40 expression was analyzed by Northern blot and polymerase chain reaction. **YKL**-40 secretion into the media was determined by enzyme-linked immunosorbent assay. Results. **YKL**-40 production increased to very high levels during the early phase of **chondrocyte** monolayer culture and in normal cartilage explant cultures as a response to tissue injury. Spontaneous **YKL**-40 release was higher in OA than in normal cartilage explant cultures. In **chondrocyte** monolayer cultures, interleukin-1.beta. (IL-1.beta.) and transforming growth factor .beta. (TGF.beta.) decreased the levels of secreted **YKL**-40, and this was associated with a reduction in **YKL**-40 messenger RNA levels. IL-1.beta., but not TGF.beta., reduced **YKL**-40 production in cartilage explant cultures. Media from explants treated with cycloheximide had no detectable **YKL**-40, suggesting that the released protein was newly synthesized. Immunofluorescence microscopy showed **YKL**-40 staining in the Golgi system of the **chondrocytes**, but **YKL**-40 could not be detected in the extracellular matrix. Conclusion. The spontaneous increase in the production of **YKL**-40 in the early phase of culture appears to represent a cellular response to changes in the extracellular matrix environment. This, coupled with the profound suppressive effects of IL-1.beta. and TGF.beta. on **YKL**-40 production, identifies a novel regulatory pattern for this major **chondrocyte**-derived protein.

L5 ANSWER 2 OF 21 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2001682113 MEDLINE
DOCUMENT NUMBER: 21584364 PubMed ID: 11727845
TITLE: Serum **YKL**-40 concentrations in patients with early rheumatoid **arthritis**: relation to joint destruction.
AUTHOR: Johansen J S; Kirwan J R; Price P A; Sharif M
CORPORATE SOURCE: Department of Rheumatology, Hvidovre Hospital, University of Copenhagen, Denmark.. julia.johansen@post3.tele.dk
SOURCE: SCANDINAVIAN JOURNAL OF RHEUMATOLOGY, (2001) 30 (5) 297-304.
Journal code: 0321213. ISSN: 0300-9742.
PUB. COUNTRY: Norway
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20011203
Last Updated on STN: 20020123
Entered Medline: 20011211
AB OBJECTIVE: **YKL**-40 is a secretory glycoprotein of **chondrocytes**, synovial cells, macrophages, and neutrophils. The aims were to determine serum **YKL**-40 in patients with early rheumatoid **arthritis** (**RA**) and seek associations with early joint erosions. METHODS: **YKL**-40 was measured by ELISA in serum samples collected every three month for 36 months from patients with

early **RA**. The patients were treated with DMARDs and some were allocated to additional prednisolone. RESULTS: Serum **YKL**-40 was higher in **RA** patients compared with controls (98 vs. 42 microg/l, p<0.001). The mean serum **YKL**-40 during the study correlated with the progression in Larsen score (Pearson's test: p=0.004). Patients with a persistently high serum **YKL**-40 had larger progression in Larsen score compared with patients with normal serum **YKL**-40 (median progression: 7 vs. 0, p=0.003). CONCLUSION: These data suggest that elevated serum **YKL**-40 is related to progression in joint destruction in early **RA** patients.

L5 ANSWER 3 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:562025 BIOSIS
DOCUMENT NUMBER: PREV200100562025
TITLE: **YKL**-40 gene expression is inhibited by cyclosporin A in human osteoblast-like cell line MG63.
AUTHOR(S): Musacchio, E. (1); Valvason, C. (1); Pozzuoli, A. (1); Priante, G. (1); Punzi, L. (1); Netto, F. S. (1); Sartori, L. (1)
CORPORATE SOURCE: (1) Department of Medical and Surgical Sciences, University of Padova, Padova Italy
SOURCE: Journal of Bone and Mineral Research, (September, 2001) Vol. 16, No. Suppl. 1, pp. S261. print.
Meeting Info.: Twenty-Third Annual Meeting of the American Society for Bone and Mineral Research Phoenix, Arizona, USA October 12-16, 2001
ISSN: 0884-0431.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L5 ANSWER 4 OF 21 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2001325116 MEDLINE
DOCUMENT NUMBER: 21198242 PubMed ID: 11300743
TITLE: Studies on **YKL**-40 in knee joints of patients with rheumatoid **arthritis** and osteoarthritis. Involvement of **YKL**-40 in the joint pathology.
AUTHOR: Volck B; Johansen J S; Stoltenberg M; Garbarsch C; Price P A; Ostergaard M; Ostergaard K; Lovgreen-Nielsen P; Sonne-Holm S; Lorenzen I
CORPORATE SOURCE: Department of Rheumatology, Hvidovre Hospital, University of Copenhagen, Denmark.. b.volck@dadlnet.dk
SOURCE: OSTEOARTHRITIS AND CARTILAGE, (2001 Apr) 9 (3) 203-14. Journal code: 9305697. ISSN: 1063-4584.
PUB. COUNTRY: England: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010611
Last Updated on STN: 20010821
Entered Medline: 20010607
AB OBJECTIVE: The presence of **YKL**-40 (human cartilage glycoprotein 39) in synovium, cartilage and synovial fluid (SF) from knee joints of patients with rheumatoid **arthritis** and osteoarthritis (OA) were related to histopathological changes in synovium and cartilage and to serum **YKL**-40 and other biochemical markers. METHODS: The localization of **YKL**-40 in synovium and cartilage was determined by immunohistochemistry. Synovial inflammation was estimated histologically and by magnetic resonance imaging (MRI). Biochemical markers of inflammation, neutrophil activation and cartilage metabolism were analysed. **YKL**-40 concentrations in serum and SF were determined by RIA and ELISA. RESULTS: In the synovium **YKL**-40 positive cells were found in lining and stromal cells (macrophages) and the number of **YKL**-40 positive cells was related to the degree of synovitis. In arthritic cartilage, **YKL**-40 was located to **chondrocytes**. **YKL**-40 levels in SF were higher in **RA** patients with moderate/severe or none/slight synovitis of the knee joint compared to OA patients with moderate/severe or none/slight synovitis. SF **YKL**-40 correlated with the synovial membrane and the joint effusion volumes determined by magnetic resonance imaging (MRI) and with other biochemical markers of intercellular matrix metabolism. SF **YKL**-40 was higher than serum **YKL**-40, and a relationship existed between the **YKL**-40 levels in SF and serum. Intraarticular glucocorticoid injection was followed by clinical remission and a decrease in serum **YKL**-40, which increased again at clinical relapse. CONCLUSIONS: **YKL**-40 in SF is derived from cells in the inflamed synovium, **chondrocytes** and SF neutrophils. Joint derived **YKL**-40 influences serum **YKL**-40. **YKL**-40 may be involved in the pathophysiology of the arthritic processes and reflect local disease activity.

L5 ANSWER 5 OF 21 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 2001474387 MEDLINE
DOCUMENT NUMBER: 21408823 PubMed ID: 11518039
TITLE: Increased level of **YKL**-40 in sera from patients with early rheumatoid **arthritis**: a new marker for disease activity.
AUTHOR: Peltomaa R; Paimela L; Harvey S; Helve T; Leirisalo-Repo M
CORPORATE SOURCE: Department of Medicine, Helsinki University Central Hospital, Finland.. Ritva.Peltomaa@hus.fi
SOURCE: RHEUMATOLOGY INTERNATIONAL, (2001 Jul) 20 (5) 192-6. Journal code: 8206885. ISSN: 0172-8172.
PUB. COUNTRY: Germany: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200201
ENTRY DATE: Entered STN: 20010827
Last Updated on STN: 20020125
Entered Medline: 20020108
AB **YKL**-40 is a newly discovered major secretory protein of human **chondrocytes** and synoviocytes. We measured serum levels of **YKL**-40 in 52 patients with early onset rheumatoid **arthritis** (**RA**) by enzyme-linked immunosorbent assay (ELISA) during a 2-year prospective follow-up, correlating values with laboratory and clinical variables and radiographic progression. Levels at baseline before antirheumatic therapy were significantly higher in patients than in healthy controls. The levels of **YKL**-40 correlated with laboratory and clinical markers of disease activity both at baseline and during follow-up. Baseline **YKL**-40 values correlated with baseline Larsen scores but did not predict radiographic

progression. Baseline and mean **YKL**-40 values did not differ between fast and slow radiological progressions. Mean **YKL**-40 levels correlated with the number of swollen joints but were not predictors of radiographic progression. These results suggest that in early **RA**, serum **YKL**-40 is an inflammatory marker correlating with disease activity. However, its levels do not predict clinical course or radiographic progression.

L5 ANSWER 6 OF 21 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 2001124047 MEDLINE
DOCUMENT NUMBER: 20566402 PubMed ID: 11114282
TITLE: Recognition of **YKL**-39, a human cartilage related protein, as a target antigen in patients with rheumatoid **arthritis**.
AUTHOR: Sekine T; Masuko-Hongo K; Matsui T; Asahara H; Takigawa M; Nishioka K; Kato T
CORPORATE SOURCE: Rheumatology, Immunology and Genetics Programme, Institute of Medical Science, St Marianna University, School of Medicine 2-16-1, Sugao, Miyamae-ku, Kawasaki, Kanagawa, 216-8512, Japan.
SOURCE: ANNALS OF THE RHEUMATIC DISEASES, (2001 Jan) 60 (1) 49-54. Journal code: 0372355. ISSN: 0003-4967.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010222

AB OBJECTIVE: To investigate whether **autoimmunity** to **YKL**-39, a recently cloned cartilage protein, occurs in patients with rheumatoid **arthritis** (**RA**). METHODS: Autoantibody to **YKL**-39 was assayed by enzyme linked immunosorbent assay (ELISA) and western blotting in serum samples from patients with **RA**, systemic lupus erythematosus (SLE), and healthy donors, using recombinant **YKL**-39 protein. This reactivity was compared with that against a **YKL**-39 homologue, **YKL**-40 (human cartilage gp-39/chondrex), which has been reported to be an autoantigen in **RA**. RESULTS: Autoantibody to **YKL**-39 was detected in seven of 87 patients with **RA** (8%), but not in serum samples from patients with SLE or healthy donors. **YKL**-40 reactivity was found in only one of 87 **RA** serum samples (1%), with no cross reactivity to **YKL**-39. CONCLUSION: The existence of anti-**YKL**-39 antibody in a subset of patients with **RA** is reported here for the first time. Further, it was shown that the immune response to **YKL**-39 was independent of that to **YKL**-40. Clarification of the antibody and T cell responses to autoantigens derived from **chondrocyte**, cartilage, or other joint components may lead to a better understanding of the pathophysiology of joint destruction in patients with **RA**.

L5 ANSWER 7 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2000:316680 BIOSIS
DOCUMENT NUMBER: PREV200000316680
TITLE: **YKL**-40 and graft rejection.
AUTHOR(S): Fiore, Carmelo E. (1); Pennisi, Pietra (1); Tamborino, Corrado
CORPORATE SOURCE: (1) Department of Internal Medicine, University of Catania, Catania Italy
SOURCE: American Journal of Medicine, (June 1, 2000) Vol. 108, No. 8, pp. 688-689. print.
ISSN: 0002-9343.
DOCUMENT TYPE: Letter
LANGUAGE: English
SUMMARY LANGUAGE: English

L5 ANSWER 8 OF 21 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:227864 CAPLUS
DOCUMENT NUMBER: 132:264105
TITLE: **YKL**-40 as a marker and prognostic indicator for cancers
INVENTOR(S): Price, Paul A.; Johansen, Julia S.
PATENT ASSIGNEE(S): The Regents of the University of California, USA
SOURCE: PCT Int. Appl., 111 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000019206	A1	20000406	WO 1999-US22615	19990929
W: AU, CA, JP, KR, NO				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9962757	A1	20000417	AU 1999-62757	19990929
EP 1112497	A1	20010704	EP 1999-950002	19990929
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1998-164862 A 19981001
WO 1999-US22615 W 19990929

AB This invention provides methods for detecting cancers and for evaluating the prognosis of cancer patients. In particular, the methods of this invention utilize **YKL**-40 as a marker for the presence or absence of a cancer and for the prognosis (e.g. likelihood of recurrence) of a cancer. Elevated levels of **YKL**-40 are indicative of the presence of a cancer in undiagnosed subjects and indicate likely recurrence of the cancer in subjects diagnosed as having a cancer.
REFERENCE COUNT: 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 9 OF 21 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 2001023517 MEDLINE
DOCUMENT NUMBER: 20460900 PubMed ID: 11005786
TITLE: Serum levels of **YKL**-40 and C reactive protein in patients with hip osteoarthritis and healthy subjects: a cross sectional study.
AUTHOR: Conrozier T; Carlier M C; Mathieu P; Colson F; Debard A L; Richard S; Favret H; Bienvenu J; Vignon E
CORPORATE SOURCE: Department of Rheumatology, Centre Hospitalier Lyon-Sud, Pierre-Benite, France.. th.conrozier@wanadoo,fr

SOURCE: ANNALS OF THE RHEUMATIC DISEASES, (2000 Oct) 59 (10) 828-31.
Journal code: 0372355. ISSN: 0003-4967.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001106

AB BACKGROUND: **YKL**-40 is a 40 kDa glycoprotein secreted by **chondrocytes** and synoviocytes. It has been suggested that it is a surrogate marker of synovial inflammation and joint destruction in rheumatoid **arthritis** (**RA**) and osteoarthritis (OA) and related to C reactive protein (CRP) serum levels in **RA**. OBJECTIVE: To study serum levels of **YKL**-40 in patients with hip OA and its relation with CRP. METHODS: **YKL**-40 and CRP were assayed in serum samples from 45 patients (24 women, 21 men, mean age 65) with symptomatic OA of the hip and 33 healthy controls. **YKL**-40 was assayed by immunoassay and CRP by ultrasensitive immunonephelometry. OA severity was assessed by the measurement of joint space width with a computer analysis system of digitised hip radiographs. Statistical analysis was performed to determine correlations between serum markers and radiological joint space width. RESULTS: The mean (standard error) **YKL**-40 level was 90.3 (8.2) ng/ml in patients with hip OA and 66.9 (8.2) ng/ml in controls (p=0.03). The mean CRP level was 2.93 (3.03) mg/l in OA and 1.40 (1.61) mg/l in controls (p=0.006). The serum levels of **YKL**-40 and CRP increased with age and were significantly correlated (Spearman test: r(s)=0.42, p=0.005) in patients but not in controls. Neither **YKL**-40 nor CRP correlated with radiographic joint space width. CONCLUSIONS: Serum **YKL**-40 was significantly increased in patients with hip OA. The correlation between **YKL** -40 and CRP suggests that **YKL**-40 may be a marker of joint inflammation in OA. Longitudinal studies are required to assess the usefulness of **YKL**-40 in the monitoring of patients with hip OA.

L5 ANSWER 10 OF 21 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 2000400313 MEDLINE
DOCUMENT NUMBER: 20334213 PubMed ID: 10873965
TITLE: Raised human cartilage glycoprotein-39 plasma levels in patients with rheumatoid **arthritis** and other inflammatory conditions.
AUTHOR: Vos K; Steenbakkers P; Miltenburg A M; Bos E; van Den Heuvel M W; van Hogezand R A; de Vries R R; Breedveld F C; Boots A M
CORPORATE SOURCE: Department of Rheumatology, LUMC, Leiden, The Netherlands.. kvos@rheumatology.azl.nl
SOURCE: ANNALS OF THE RHEUMATIC DISEASES, (2000 Jul) 59 (7) 544-8.
Journal code: 0372355. ISSN: 0003-4967.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000824
Last Updated on STN: 20000824
Entered Medline: 20000816

AB OBJECTIVE: To evaluate plasma human cartilage glycoprotein (HC gp-39) as a possible marker for the presence and/or activity of rheumatoid **arthritis** (**RA**) and other inflammatory conditions. BACKGROUND: HC gp-39 is a secretory product of **chondrocytes**, synovial cells, macrophages, and neutrophils. HC gp-39, also described as **YKL**-40, was found to be a marker of joint disease and tissue injury in **RA** and various other diseases. METHODS: Levels of HC gp-39 were determined by a sandwich enzyme linked immunosorbent assay (ELISA) in 47 patients with **RA**, 47 with osteoarthritis (OA), 24 with systemic lupus erythematosus (SLE), 24 with inflammatory bowel disease (IBD), and in 47 healthy controls. A disease activity score was assessed in the patients with **RA**, SLE, and IBD. RESULTS: The plasma level of HC gp-39 in the **RA** patient group was significantly higher than in the other patient groups and healthy controls. The level in patients with OA, SLE, and IBD was also significantly higher than the HC gp-39 level found in the healthy control group. HC gp-39 levels in patients with **RA** correlated positively with the ESR and IgM rheumatoid factor level but not with other variables of disease activity. In the patients with SLE and IBD no correlation was found with the disease activity score. CONCLUSION: The plasma level of HC gp-39 is increased in inflammatory conditions with and without joint disease (SLE, IBD, OA, and **RA**). Thus increased levels of HC gp-39 do not only reflect joint disease but also reflect inflammation or tissue degradation in various conditions. Notably, the highest level of HC gp-39 was found in patients with **RA**. Only in the **RA** patient group was a correlation between HC gp-39 plasma levels and some laboratory variables of disease activity found.

L5 ANSWER 11 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2000:185155 BIOSIS
DOCUMENT NUMBER: PREV200000185155
TITLE: Serum chondrex values in knee osteoarthritis (OA). The effect of arthroscopy.
AUTHOR(S): Maciel, S. B.; Scheinberg, M. A. (1)
CORPORATE SOURCE: (1) 26 Dr Martinico Prado, Sao Paulo, SP, 01224-010 Brazil
SOURCE: Clinical Rheumatology, (2000) Vol. 19, No. 1, pp. 76-77. ISSN: 0770-3198.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English

L5 ANSWER 12 OF 21 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 2000488238 MEDLINE
DOCUMENT NUMBER: 20492231 PubMed ID: 11037631
TITLE: [Biochemical markers of bone turnover and **YKL** 40 in ankylosing spondylitis. Relation to disease activity]. Marcatori biochimici di turnover osseo e **YKL** 40 nella spondilite anchilosante. Rapporto con l'attivita di malattia.
AUTHOR: D'Amore M; Germinario G; D'Amore S; Scagliusi P
CORPORATE SOURCE: Dipartimento di Medicina Interna e di Medicina Pubblica, Universita degli Studi, Bari.
SOURCE: MINERVA MEDICA, (2000 Mar-Apr) 91 (3-4) 59-68.
Journal code: 0400732. ISSN: 0026-4806.
PUB. COUNTRY: Italy

Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Italian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001128

AB BACKGROUND: **YKL**-40 is a glycoprotein produced by **chondrocytes** and synovial cells. The plasmatic levels of this metabolite increase in some pathologies such as rheumatoid **arthritis** and osteoarthrosis, so much so that it can be considered an effective marker of disease activity and in the therapeutic monitoring of these diseases. It has been interesting to dose a group of both male and female subjects affected by seronegative spondylarthritis, comparing this parameter with the disease activity indexes and with the bone turnover markers. METHODS: The study has been carried out on 48 subjects (26 males and 22 females) between 17 and 68 years affected by spondylarthritis, diagnosed in conformity with ARA standards. None of the patients carried out basic treatment or by glycocorticoids, and 22 patients took FANS when required. In these subjects the disease activity markers (VES, PCR, fibrinogen, mucoprotein) and some of the classic bone remodelling markers (blood calcium and phosphates, calciuria, phosphaturia, Ca++, Ntx, osteocalcine, bone isoenzyme of alkaline phosphatase, hydroxyproline, procollagen and **YKL**-40) were dosed. RESULTS: The comparison between different parameters pointed out that the highest values are obtained in subjects of most advanced age with the highest phlogosis indexes, without any correlation with sex. The quite interesting comparison shows a correlation between the bone remodelling indexes and **YKL**-40, being particularly remarkable when the disease is more aggressive or during relapse. CONCLUSIONS: It is then possible to confirm that, though preliminary, these data may suggest evaluations on wider case histories to research **YKL**-40 as a surgical monitoring marker in seronegative poliarthritis.

L5 ANSWER 13 OF 21 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 1999390575 MEDLINE
DOCUMENT NUMBER: 99390575 PubMed ID: 10461474
TITLE: Serum **YKL**-40 concentrations in patients with rheumatoid **arthritis**: relation to disease activity.
AUTHOR: Johansen J S; Stoltenberg M; Hansen M; Florescu A; Horslev-Petersen K; Lorenzen I; Price P A
CORPORATE SOURCE: Department of Rheumatology, Hvidovre Hospital, Denmark.
SOURCE: RHEUMATOLOGY, (1999 Jul) 38 (7) 618-26.
Journal code: 100883501. ISSN: 1462-0324.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199909
ENTRY DATE: Entered STN: 19990921
Last Updated on STN: 19990921
Entered Medline: 19990908

AB OBJECTIVE: **YKL**-40, also called human cartilage glycoprotein-39, is secreted by **chondrocytes**, synovial cells, macrophages and neutrophils. Studies have shown that **YKL**-40 is an autoantigen in rheumatoid **arthritis** (**RA**). We evaluated whether serum **YKL**-40 was related to disease activity in patients with **RA**. METHODS: Serum **YKL**-40 was determined by radioimmunoassay in 156 patients with **RA** during a 1 yr longitudinal study. RESULTS: Serum **YKL**-40 was increased in 54% of the patients with clinically active disease. Patients with clinically active disease initially who became inactive after 12 months had a significant decrease in serum **YKL**-40 (-30%, $P < 0.002$) and patients who changed from inactive to active disease had an increase in serum **YKL**-40. Patients who remained active had unchanged serum **YKL**-40 during the study. Serum **YKL**-40 decreased rapidly (-24% after 7 days, $P < 0.01$) during prednisolone therapy, and more slowly in patients treated with methotrexate only (-15% after 60 days, $P < 0.01$). Patients with early **RA** (disease duration < 3 yr, n = 50) and a persistently elevated serum **YKL**-40 were at risk of radiological disease progression as determined by Larsen score. CONCLUSION: Serum **YKL**-40 varies according to disease activity in **RA**, but provides in some respect information different from conventional markers. Our previous studies are consistent with a local release of **YKL**-40 in the arthritic joint followed by a secondary increase in serum **YKL**-40. **YKL**-40 may prove to be a new tool for the study of disease activity and pathophysiology of **RA**.

L5 ANSWER 14 OF 21 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 1999249149 EMBASE
TITLE: Nitric oxide alters **chondrocyte** function by disrupting cytoskeletal signaling complexes.
AUTHOR: Clancy R.
CORPORATE SOURCE: R. Clancy, Department of Rheumatology, Hospital for Joint Diseases, NYU School of Medicine, New York, NY, United States
SOURCE: Osteoarthritis and Cartilage, (1999) 7/4 (399-400).
Refs: 10
ISSN: 1063-4584 CODEN: OSCAEO
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 029 Clinical Biochemistry
031 Arthritis and Rheumatism
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Components of osteoarthritis include increases in pericellular fibronectin and in **chondrocyte** .beta.1 integrin expression. Events which follow ligation of fibronectin to its **chondrocyte**-receptor, the integrin .alpha.5.beta.1 include an assembly of a subplasmalemmal actin/rho A/focal adhesion kinase signaling complex. In addition, nitric oxide (NO), a potential mediator of cartilage pathophysiology disrupts the cytoskeletal signaling complex associated with integrin signaling. In these studies, we examined the relationship among integrin signaling, biosynthesis of S-35 sulfate containing proteoglycans and release of **YKL**-40 (a secretory glycoprotein) by comparing cell responses using cells plated on a fibronectin-coated or polyHEME coated surfaces. We report that the release of proteoglycan and glycoprotein require anchorage dependent signals by integrin costimulation. NO which disrupts the integrin signaling complex attenuates both cell responses. Taken together NO may serve as a nonspecific 'brake' to prevent anabolic and catabolic injury responses.

L5 ANSWER 15 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:537094 BIOSIS
DOCUMENT NUMBER: PREV199900537094
TITLE: A novel **arthritis** model mice by secretory protein of articular **chondrocytes**, **YKL**-39.
AUTHOR(S): Sakata, Masahiro (1); Masuko-Hongo, Kayo; Tsuruha, Jun-ichiro; Nakamura, Hiroshi; Sekine, Taichi; Yoshino, Shin-ichi; Takigawa, Masaharu; Kato, Tomohiro; Nishioka, Kusuki
CORPORATE SOURCE: (1) Kawasaki Japan
SOURCE: Arthritis & Rheumatism, (Sept., 1999) Vol. 42, No. 9 SUPPL., pp. S257.
Meeting Info.: 63rd Annual Scientific Meeting of the American College of Rheumatology and the 34th Annual Scientific Meeting of the Association of Rheumatology Health Professionals Boston, Massachusetts, USA November 13-17, 1999
ISSN: 0004-3591.
DOCUMENT TYPE: Conference
LANGUAGE: English

L5 ANSWER 16 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:537080 BIOSIS
DOCUMENT NUMBER: PREV199900537080
TITLE: Regulation of **YKL**-40 production by human articular **chondrocytes**.
AUTHOR(S): Johansen, Julia S. (1); Olee, Tsaiwei (1); Price, Paul A. (1); Ochs, Robert L. (1); Hashimoto, Sanshiro (1); Lotz, Martin (1)
CORPORATE SOURCE: (1) La Jolla, CA USA
SOURCE: Arthritis & Rheumatism, (Sept., 1999) Vol. 42, No. 9 SUPPL., pp. S255.
Meeting Info.: 63rd Annual Scientific Meeting of the American College of Rheumatology and the 34th Annual Scientific Meeting of the Association of Rheumatology Health Professionals Boston, Massachusetts, USA November 13-17, 1999
ISSN: 0004-3591.
DOCUMENT TYPE: Conference
LANGUAGE: English

L5 ANSWER 17 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:535492 BIOSIS
DOCUMENT NUMBER: PREV199900535492
TITLE: Serum **YKL**-40, a **chondrocyte** derived protein is reduced by infliximab (anti-TNF) therapy in patients with rheumatoid **arthritis**.
AUTHOR(S): Charles, P. J. (1); Maini, R. N. (1)
CORPORATE SOURCE: (1) London UK
SOURCE: Arthritis & Rheumatism, (Sept., 1999) Vol. 42, No. 9 SUPPL., pp. S236.
Meeting Info.: 63rd Annual Scientific Meeting of the American College of Rheumatology and the 34th Annual Scientific Meeting of the Association of Rheumatology Health Professionals Boston, Massachusetts, USA November 13-17, 1999
ISSN: 0004-3591.
DOCUMENT TYPE: Conference
LANGUAGE: English

L5 ANSWER 18 OF 21 MEDLINE DUPLICATE 9
ACCESSION NUMBER: 1999308547 MEDLINE
DOCUMENT NUMBER: 99308547 PubMed ID: 10380840
TITLE: The distribution of **YKL**-40 in osteoarthritic and normal human articular cartilage.
AUTHOR: Volck B; Ostergaard K; Johansen J S; Garbarsch C; Price P A
CORPORATE SOURCE: Department of Rheumatology, Hvidovre Hospital, Denmark.
SOURCE: SCANDINAVIAN JOURNAL OF RHEUMATOLOGY, (1999) 28 (3) 171-9.
Journal code: 0321213. ISSN: 0300-9742.
PUB. COUNTRY: Norway
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 19990714
Last Updated on STN: 19990714
Entered Medline: 19990629
AB **YKL**-40, also called human cartilage glycoprotein-39, is a major secretory protein of human **chondrocytes** in cell culture. **YKL**-40 mRNA is expressed by cartilage from patients with rheumatoid **arthritis**, but is not detectable in normal human cartilage. The aim was to investigate the distribution of **YKL**-40 in osteoarthritic (n=9) and macroscopically normal (n=5) human articular cartilage, collected from 12 pre-selected areas of the femoral head, to discover a potential role for **YKL**-40 in cartilage remodelling in osteoarthritis. Immunohistochemical analysis showed that **YKL**-40 staining was found in **chondrocytes** of osteoarthritic cartilage mainly in the superficial and middle zone of the cartilage rather than the deep zone. There was a tendency for high number of **YKL**-40 positive **chondrocytes** in areas of the femoral head with a considerable biomechanical load. The number of **chondrocytes** with a positive staining for **YKL**-40 was in general low in normal cartilage. The present findings, together with previous observations, suggests that **YKL**-40 may be of importance in cartilage remodelling/degradation of osteoarthritic joints.

L5 ANSWER 19 OF 21 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:170374 CAPLUS
DOCUMENT NUMBER: 128:260523
TITLE: Chondrex: new marker of joint disease
AUTHOR(S): Harvey, Sheryl; Weisman, Michael; O'Dell, James; Scott, Tonya; Krusemeier, Mindy; Visor, Jill; Swindlehurst, Cathy
CORPORATE SOURCE: Novadex, Inc, San Diego, CA, 92121, USA
SOURCE: Clinical Chemistry (Washington, D. C.) (1998), 44(3), 509-516
CODEN: CLCHAU; ISSN: 0009-9147
PUBLISHER: American Association for Clinical Chemistry
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Chondrex, a major secretory protein of human **chondrocytes** and synovial fibroblasts, is increased in serum of patients with joint and cartilage disease. We have developed a sandwich-type ELISA for

quantifying chondrex in serum. The interassay CVs were 2.8-3.7% and the av. within-run and total CVs were 3.6% and 5.4%, resp. The limit of detectability by linear diln. was 20 .mu.g/L, recovery upon diln. was 102% .+-. 5%, and anal. recovery (of added analyte) was 98% .+-. 11%. The ref. interval (central 90% interval) for chondrex in healthy adults was 25-95 .mu.g/L. Chondrex values for patients with active rheumatoid **arthritis** or osteoarthritis were significantly greater than in healthy adults, inactive rheumatoid **arthritis** patients, and diabetes patients (P <0.05). In patients treated with disease-modifying antirheumatic drug therapy, decreasing chondrex values reflected the clin. improvement obsd. in responders, whereas the values were maintained or increased in nonresponders. In conclusion, chondrex may be a useful marker in the clin. investigation of **arthritis**.

L5 ANSWER 20 OF 21 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 1998349472 MEDLINE
DOCUMENT NUMBER: 98349472 PubMed ID: 9686683
TITLE: **YKL**-40, a mammalian member of the chitinase family, is a matrix protein of specific granules in human neutrophils.
AUTHOR: Volck B; Price P A; Johansen J S; Sorensen O; Benfield T L; Nielsen H J; Calafat J; Borregaard N
CORPORATE SOURCE: Department of Rheumatology, Hvidovre Hospital, University of Copenhagen, Denmark.
SOURCE: PROCEEDINGS OF THE ASSOCIATION OF AMERICAN PHYSICIANS, (1998 Jul-Aug) 110 (4) 351-60.
Journal code: 9514310. ISSN: 1081-650X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199904
ENTRY DATE: Entered STN: 19990426
Last Updated on STN: 19990426
Entered Medline: 19990413

AB **YKL**-40, also called human cartilage glycoprotein-39 (HC gp-39), is a member of family 18 glycosyl hydrolases. **YKL**-40 is secreted by **chondrocytes**, synovial cells, and macrophages, and recently it has been reported that **YKL**-40 has a role as an autoantigen in rheumatoid **arthritis** (**RA**). The function of **YKL**-40 is unknown, but the pattern of its expression in normal and disease states suggests that it could function in remodeling or degradation of the extracellular matrix. High levels of **YKL**-40 are found in synovial fluid from patients with active **RA**. Neutrophils are abundant in synovial fluid of patients with **RA**, and the cells are assumed to play a role in joint destruction in that disorder. Therefore, we examined whether neutrophils are a source of **YKL**-40. **YKL**-40 was found to colocalize and comobilize with lactoferrin (the most abundant protein of specific granules) but not with gelatinase in subcellular fractionation studies on stimulated and unstimulated neutrophils. Double-labeling immunoelectron microscopy confirmed the colocalization of **YKL**-40 and lactoferrin in specific granules of neutrophils. Immunohistochemistry on bone marrow cells showed that neutrophil precursors begin to synthesize **YKL**-40 at the myelocyte-metamyelocyte stage, the stage of maturation at which other specific granule proteins are formed. Assuming that **YKL**-40 has a role as an autoantigen in **RA** by inducing T cell-mediated **autoimmune** response, **YKL**-40 released from neutrophils in the inflamed joint could be essential for this response. In **RA** and other inflammatory diseases, **YKL**-40 released from specific granules of neutrophils may be involved in tissue remodeling or degradation.

L5 ANSWER 21 OF 21 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 93334909 EMBASE
DOCUMENT NUMBER: 1993334909
TITLE: A new biochemical marker for joint injury. Analysis of **YKL** 40 in serum and synovial fluid.
AUTHOR: Johansen J.S.; Jensen H.S.; Price P.A.
CORPORATE SOURCE: Dept of Med, Div of Rheumatology 232, University of Copenhagen, Hvidore Hospital, Kettegard Alle 30,Hvidore DK-2650, Denmark
SOURCE: British Journal of Rheumatology, (1993) 32/11 (949-955).
ISSN: 0263-7103 CODEN: BJRHDF
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 031 Arthritis and Rheumatism
LANGUAGE: English
SUMMARY LANGUAGE: English

AB We report the development of the first radioimmunoassay for **YKL**-40, a M(r) = 40 kDa protein which is secreted at high levels by human synovial cells and articular cartilage **chondrocytes**, and by the human osteosarcoma cell line MG63. This assay uses **YKL**-40 purified from the conditioned medium of MG63 cells as standard and tracer, and as antigen for immunizing rabbits. With this assay we have discovered high levels of **YKL**-40 antigen in serum and SF. The molecular weight of serum and SF **YKL**-40 is identical to purified **YKL**-40. To evaluate the possible utility of **YKL**-40 in the assessment of joint disease, we measured **YKL**-40 in serum and SF of 49 patients with various forms of inflammatory and degenerative joint disease and in the serum of 50 normal adults. The **YKL**-40 level in serum was significantly higher (P<0.001) in the patients compared to the normal adults, but there was no difference in serum **YKL**-40 between the patients with inflammatory joint diseases and OA. The SF levels of **YKL**-40 were 15-fold higher than serum levels and there was a significant correlation (r = 0.55, P<0.001) between **YKL**-40 concentration in SF and serum. Although the tissue distribution of **YKL**-40 secretion is presently unknown, these observations suggest that a major portion of serum **YKL**-40 in fact arises from the joint. Serum and SF **YKL** 40 levels are correlated significantly (P<0.05-P<0.001) with other indices of joint disease: serum CRP SF IL-6, and the elastolytic activity of monocytes/macrophages in SF. Serum **YKL**-40 also correlated with serum PIIINP and elastolytic activity of blood monocytes/macrophages. These studies indicate that serum and SF **YKL**-40 levels reflect joint disease and a **YKL**-40 determination may therefore be useful in the evaluation of connective tissue injury and repair in patients with inflammatory or degenerative rheumatic diseases. Future studies will be needed in order to assess the physiologic significance of elevated **YKL**-40 levels in patients with rheumatoid diseases.

=> s ykl (1N) 39

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L6          19 YKL (1N) 39

=> dup rem l6
PROCESSING COMPLETED FOR L6
L7          10 DUP REM L6 (9 DUPLICATES REMOVED)

=> dis l7 1-10 ibib abs

L7   ANSWER 1 OF 10  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER:   2002:261273  BIOSIS
DOCUMENT NUMBER:    PREV200200261273
TITLE:              Analysis of chondrex (YKL-40, HC gp-39) in the
                    cerebrospinal fluid of patients with spine disease.
AUTHOR(S):          Tsuji, Taichi (1); Matsuyama, Yukihiro; Natsume, Naoki;
                    Hasegawa, Yukiharu; Kondo, Seiji; Kawakami, Hiroshi;
                    Yoshihara, Hisatake; Iwata, Hisashi
CORPORATE SOURCE:   (1) Department of Orthopaedic Surgery, Nagoya University
                    School of Medicine, 65 Tsuruma-cho, Showa-ku, Nagoya,
                    466-8550: tsuji-t@med.nagoya-u.ac.jp Japan
SOURCE:             Spine, (April 1, 2002) Vol. 27, No. 7, pp. 732-735.
                    http://www.spinejournal.com/. print.
                    ISSN: 0362-2436.
DOCUMENT TYPE:      Article
LANGUAGE:           English
AB   Study Design: The expression of chondrex (YKL-40, HC gp-39) was measured
     in the cerebrospinal fluid of from patients with spine diseases.
     Objectives: To quantify the levels of chondrex in human cerebrospinal
     fluid, and to clarify the nature of its expression. Summary of Background
     Data: Chondrex is a newly discovered 40-kDa glycoprotein identified
     originally in the whey secretions of nonlactating cows. It is secreted by
     a human osteosarcoma cell line, human articular cartilage chondrocytes,
     and human fibroblasts. However, the function of chondrex in chondrogenesis
     is unknown, and the expression of chondrex in human cerebrospinal fluid
     has never been reported. Methods: The concentration of chondrex in human
     cerebrospinal fluid was measured by sandwich immunoassay with antihuman
     chondrex antibodies. Cerebrospinal fluid samples were collected from two
     groups of patients. Group 1, the control group, consisted of 34 trauma
     patients. Group 2 consisted of 130 patients with spine diseases: 29 with
     cervical spondylotic myelopathy, 30 with lumbar disc herniation, 35 with
     lumbar canal stenosis, and 36 with scoliosis. All values are expressed as
     the mean+-standard deviation. Results: The concentration of chondrex in
     Group 1 (control group) was 113.8+-48.3 ng/mL. The concentrations of
     chondrex in Group 2 were 245.3+-107.2 ng/mL in cervical myelopathy,
     143.2+-53.6 ng/mL in lumbar disc herniation, 241.5+-77.2 ng/mL in lumbar
     canal stenosis, and 71.4+-33.9 ng/mL in scoliosis. The concentrations of
     chondrex in cervical myelopathy, lumbar canal stenosis, and lumbar disc
     herniation were significantly higher than in the control group (P<0.05).
     Conclusions: In this study, the chondrex concentration was high in spine
     diseases causing spinal stenosis. The authors believe that chondrex is
     expressed in cerebrospinal fluid as a result of damage or stress to the
     neural structure, and that it could be a new marker for spine diseases.

L7   ANSWER 2 OF 10     MEDLINE                          DUPLICATE 1
ACCESSION NUMBER:   2001252197     MEDLINE
DOCUMENT NUMBER:    21248333   PubMed ID: 11350852
TITLE:              Cross sectional evaluation of biochemical markers of bone,
                    cartilage, and synovial tissue metabolism in patients with
                    knee osteoarthritis: relations with disease activity and
                    joint damage.
COMMENT:            Comment in: Ann Rheum Dis. 2001 Jun;60(6):545-8
AUTHOR:             Garnero P; Piperno M; Gineyts E; Christgau S; Delmas P D;
                    Vignon E
CORPORATE SOURCE:   Inserm Research Unit 403, Lyon, France..
                    patrick.garnero@synarc.com
SOURCE:             ANNALS OF THE RHEUMATIC DISEASES, (2001 Jun) 60 (6) 619-26.
                    Journal code: 0372355. ISSN: 0003-4967.
PUB. COUNTRY:       England: United Kingdom
                    (EVALUATION STUDIES)
                    Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        200106
ENTRY DATE:         Entered STN: 20010611
                    Last Updated on STN: 20010611
                    Entered Medline: 20010607
AB   OBJECTIVE: To analyse the relations between the urinary levels of type II
     collagen C-telopeptide (CTX-II) and glucosyl-galactosyl pyridinoline
     (Glc-Gal-PYD)-two newly developed biochemical markers of type II collagen
     and synovial tissue destruction respectively-disease activity and the
     severity of joint destruction in patients with knee osteoarthritis (OA).
     The clinical performance of these two new markers was compared with that
     of a panel of other established biochemical markers of connective tissue
     metabolism. METHODS: The following biochemical markers were measured in a
     group of 67 patients with knee OA (mean age 64 years, median disease
     duration eight years ) and in 67 healthy controls: for bone, serum
     osteocalcin, serum and urinary C-telopeptide of type I collagen (CTX-I);
     for cartilage, urinary CTX-II, serum cartilage oligomeric matrix protein
     (COMP), and serum human cartilage glycoprotein 39 (YKL
     -40); for synovium, urinary Glc-Gal-PYD, serum type III collagen
     N-propeptide (PIIINP), serum hyaluronic acid (HA); and for inflammation,
     serum C reactive protein. Biochemical markers were correlated with pain
     and physical function (WOMAC index) and with quantitative radiographic
     evaluation of the joint space using the posteroanterior view of the knees
     flexed at 30 degrees. RESULTS: All bone turnover markers were decreased in
     patients with knee OA compared with controls (-36%, -38%, and -52%,
     p<0.0001 for serum osteocalcin, serum CTX-I and urinary CTX-I,
     respectively). Serum COMP (+16%, p=0.0004), urinary CTX-II (+25%,
     p=0.0009), urinary Glc-Gal-PYD (+18%, p=0.028), serum PIIINP (+33%,
     p<0.0001), and serum HA (+ 233%, p<0.0001) were increased. By univariate
     analyses, increased urinary Glc-Gal-PYD (r=0.41, p=0.002) and decreased
     serum osteocalcin (r=-0.30, p=0.025) were associated with a higher total
     WOMAC index. Increased urinary CTX-II (r=-0.40, p=0.0002) and Glc-Gal-PYD
     (r=-0.30, p=0.0046) and serum PIIINP (r=-0.29, p=0.0034) were the only
     markers which correlated with joint surface area. By multivariate
     analyses, urinary Glc-Gal-PYD and CTX-II were the most important
     predictors of the WOMAC index and joint damage, respectively. CONCLUSION:
     Knee OA appears to be characterised by a systemic decrease of bone
     turnover and increased cartilage and synovial tissue turnover. CTX-II,
     Glc-Gal-PYD, and PIIINP may be useful markers of disease severity in
     patients with knee OA.

L7   ANSWER 3 OF 10  CAPLUS  COPYRIGHT 2002 ACS
ACCESSION NUMBER:        2001:315060  CAPLUS
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DOCUMENT NUMBER: 134:363566
TITLE: Development of an enzyme-linked immunoassay for the quantification of YKL-40 (cartilage gp-39) in guinea pig serum using hen egg yolk antibodies
AUTHOR(S): De Ceuninck, F.; Pastoureau, P.; Agnellet, S.; Bonnet, J.; Vanhoutte, P. M.
CORPORATE SOURCE: Division of Rheumatology, Institut de Recherches Servier, Suresnes, 92150, Fr.
SOURCE: Journal of Immunological Methods (2001), 252(1-2), 153-161
CODEN: JIMMBG; ISSN: 0022-1759
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB An indirect competition immunoassay for the quantification of YKL-40 (cartilage gp-39, Chondrex) in guinea pig serum has been developed using egg yolk antibodies (IgY). The immune response of hens to YKL-40 was verified by immunoblot analyses. Highly specific antibodies were obtained 30 days after the first injection. The ELISA was developed in 96-well microtiter plates with quadruplicate detns. for each point. The assay was based on the ability of YKL-40 present in serum to displace the binding of antibodies to the coated antigen. An inhibition mixt. contg. std. YKL-40 or guinea pig serum, dild. 1/5, and primary antibodies, dild. 1/5000, was allowed to equilibrate for 2 h at room temp. and dispensed for 16 h at 4.degree. in wells coated with 1 .mu.g/mL of YKL-40. Detection was achieved by the addn. of rabbit anti-chicken antibodies conjugated to peroxidase followed by tetramethylbenzidine. Specificity was assessed by parallelism between a diln. curve of serum and std. YKL-40. The sensitivity of detection was 10 ng/mL. Intra- and interassay coeffs. of variation were both 8.7%. The anal. recovery was 101.5.+-.5.4% (mean.+-.std. deviation (SD), n=9). The YKL-40 concn. in serum from 12 adult guinea pigs was 330.+-.216 ng/mL (mean.+-.SD) with a lower value of 164 ng/mL and an upper value of 982 ng/mL. In contrast to the rat, a diln. curve of rabbit serum gave parallelism with the guinea pig std., suggesting recognition of a similar epitope. Possible applications of the assay in the guinea pig include disease models where YKL-40 is overexpressed and could be used as a marker, i.e. osteoarthritis, rheumatoid arthritis, cancer, liver fibrosis, atherosclerosis and more generally, pathologies with increased tissue remodeling.
REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 4 OF 10 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2001124047 MEDLINE
DOCUMENT NUMBER: 20566402 PubMed ID: 11114282
TITLE: Recognition of **YKL-39**, a human cartilage related protein, as a target antigen in patients with rheumatoid arthritis.
AUTHOR: Sekine T; Masuko-Hongo K; Matsui T; Asahara H; Takigawa M; Nishioka K; Kato T
CORPORATE SOURCE: Rheumatology, Immunology and Genetics Programme, Institute of Medical Science, St Marianna University, School of Medicine 2-16-1, Sugao, Miyamae-ku, Kawasaki, Kanagawa, 216-8512, Japan.
SOURCE: ANNALS OF THE RHEUMATIC DISEASES, (2001 Jan) 60 (1) 49-54. Journal code: 0372355. ISSN: 0003-4967.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010222

AB OBJECTIVE: To investigate whether autoimmunity to **YKL-39**, a recently cloned cartilage protein, occurs in patients with rheumatoid arthritis (RA). METHODS: Autoantibody to **YKL-39** was assayed by enzyme linked immunosorbent assay (ELISA) and western blotting in serum samples from patients with RA, systemic lupus erythematosus (SLE), and healthy donors, using recombinant **YKL-39** protein. This reactivity was compared with that against a **YKL-39** homologue, **YKL**-40 (human cartilage gp-39/chondrex), which has been reported to be an autoantigen in RA. RESULTS: Autoantibody to **YKL-39** was detected in seven of 87 patients with RA (8%), but not in serum samples from patients with SLE or healthy donors. YKL-40 reactivity was found in only one of 87 RA serum samples (1%), with no cross reactivity to **YKL-39**. CONCLUSION: The existence of anti-**YKL-39** antibody in a subset of patients with RA is reported here for the first time. Further, it was shown that the immune response to **YKL-39** was independent of that to YKL-40. Clarification of the antibody and T cell responses to autoantigens derived from chondrocyte, cartilage, or other joint components may lead to a better understanding of the pathophysiology of joint destruction in patients with RA.

L7 ANSWER 5 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:537094 BIOSIS
DOCUMENT NUMBER: PREV199900537094
TITLE: A novel arthritis model mice by secretory protein of articular chondrocytes, **YKL-39**.
AUTHOR(S): Sakata, Masahiro (1); Masuko-Hongo, Kayo; Tsuruha, Jun-ichiro; Nakamura, Hiroshi; Sekine, Taichi; Yoshino, Shin-ichi; Takigawa, Masaharu; Kato, Tomohiro; Nishioka, Kusuki
CORPORATE SOURCE: (1) Kawasaki Japan
SOURCE: Arthritis & Rheumatism, (Sept., 1999) Vol. 42, No. 9 SUPPL., pp. S257.
Meeting Info.: 63rd Annual Scientific Meeting of the American College of Rheumatology and the 34th Annual Scientific Meeting of the Association of Rheumatology Health Professionals Boston, Massachusetts, USA November 13-17, 1999
ISSN: 0004-3591.
DOCUMENT TYPE: Conference
LANGUAGE: English

L7 ANSWER 6 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:537097 BIOSIS
DOCUMENT NUMBER: PREV199900537097
TITLE: Agument of articular immune responses to gp-**39** and **YKL-39** in patients with osteoarthritis.
AUTHOR(S): Tsuruha, Jun-ichiro (1); Masuko-Hongo, Kayo; Sakata,

Masahiro; Nakamura, Hiroshi; Sekine, Taichi; Yoshino, Shin-ichi; Takigawa, Masaharu; Kato, Tomohiro; Nishioka, Kusuki
CORPORATE SOURCE: (1) Kawasaki Japan
SOURCE: Arthritis & Rheumatism, (Sept., 1999) Vol. 42, No. 9 SUPPL., pp. S257.
Meeting Info.: 63rd Annual Scientific Meeting of the American College of Rheumatology and the 34th Annual Scientific Meeting of the Association of Rheumatology Health Professionals Boston, Massachusetts, USA November 13-17, 1999
ISSN: 0004-3591.
DOCUMENT TYPE: Conference
LANGUAGE: English

L7 ANSWER 7 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:432310 BIOSIS
DOCUMENT NUMBER: PREV199900432310
TITLE: Possible quantitative trait loci for serum levels of human cartilage glycoprotein-39 (YKL-40) and osteocalcin (OC) in pedigreed baboons map to human chromosomes 6 and 12.
AUTHOR(S): Mahaney, M. C. (1); Czerwinski, S. A. (1); Rogers, J. (1)
CORPORATE SOURCE: (1) Genetics, Southwest Foundation for Biomedical Research, San Antonio, TX USA
SOURCE: Journal of Bone and Mineral Research, (Sept., 1999) Vol. 14, No. SUPPL. 1, pp. S142.
Meeting Info.: Twenty-First Annual Meeting of the American Society for Bone and Mineral Research St. Louis, Missouri, USA September 30-October 4, 1999 American Society for Bone and Mineral Research
. ISSN: 0884-0431.
DOCUMENT TYPE: Conference
LANGUAGE: English

L7 ANSWER 8 OF 10 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:718004 CAPLUS
DOCUMENT NUMBER: 128:16403
TITLE: Human cartilage autoantigen glycoprotein gp-39 and proteins structurally related thereto for use in immunotherapy of autoimmune diseases
INVENTOR(S): Boots, Anna Maria Helena; Verheijden, Gijsbertus Franciscus Maria; Bos, Ebo Sybren
PATENT ASSIGNEE(S): Akzo Nobel N.V., Neth.; Boots, Anna Maria Helena; Verheijden, Gijsbertus Franciscus Maria; Bos, Ebo Sybren
SOURCE: PCT Int. Appl., 38 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9740149	A1	19971030	WO 1997-EP1903	19970415
W: AM, AU, BB, BG, BR, CA, CN, CZ, EE, GE, HU, IS, JP, KG, KP, KR, LK, LR, LT, LV, MD, MG, MN, MX, NO, NZ, PL, RO, RU, SG, SI, SK, TR, TT, UA, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 5843449	A	19981201	US 1996-634493	19960418
AU 9723869	A1	19971112	AU 1997-23869	19970415
AU 724547	B2	20000928		
EP 904369	A1	19990331	EP 1997-919370	19970415
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
BR 9708714	A	19990803	BR 1997-8714	19970415
JP 2000509265	T2	20000725	JP 1997-537477	19970415
NO 9804835	A	19981216	NO 1998-4835	19981016

PRIORITY APPLN. INFO.: US 1996-634493 A 19960418
US 1996-619645 A2 19960325
WO 1997-EP1903 W 19970415

AB The present invention relates to the use of autoantigen HC gp-39 (human cartilage glycoprotein-39), and proteins comprising an amino acid sequence which exhibits .gtoreq.50% homol. with the amino acid sequence of HC gp-39, and more particular with the amino acid sequence YKLVCYYTSWSQYREGDGSCFPDALDRFLCTHIIYSFANISND in antigen-specific treatment of articular cartilage destruction in autoimmune diseases in mammals to induce systemic tolerance of the immune system. The autoantigen HC gp-39, and the arthritogenic proteins comprising an amino acid sequence which exhibits .gtoreq.50% homol. with the amino acid sequence YKLVCYYTSWSQYREGDGSCFPDALDRFLCTHIIYSFANISND are also suitable to induce arthritis in animals, preferably mice. The arthritogenicity and cloning of bovine 39-kDa whey protein is also described. The invention furthermore relates to pharmaceutical compns. comprising said autoantigen and/or said arthritogenic proteins, a diagnostic method for the detection of autoreactive T cells in a test sample and test kits to be used in said method.

L7 ANSWER 9 OF 10 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:383815 CAPLUS
DOCUMENT NUMBER: 127:62133
TITLE: Various roles of chitinases
AUTHOR(S): Watanabe, Takeshi
CORPORATE SOURCE: Nogakubu, Niigata Daigaku, Niigata, 950-21, Japan
SOURCE: Kagaku to Seibutsu (1997), 35(6), 408-414
CODEN: KASEAA; ISSN: 0453-073X
PUBLISHER: Gakkai Shuppan Senta
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese

AB A review, with 15 refs., on occurrence, physiol. function, classification, and conformation of chitinases. Reaction mechanism of chitinases of family 18 and proteins structurally analogous to family 18 chitinases, e.g. cartilage glycoprotein gp-39 (YKL-40), oviductin, concanavalin B, narbonin. etc., are also discussed.

L7 ANSWER 10 OF 10 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 96325055 MEDLINE
DOCUMENT NUMBER: 96325055 PubMed ID: 8702629
TITLE: Isolation and sequence of a novel human chondrocyte protein related to mammalian members of the chitinase protein

family.
AUTHOR: Hu B; Trinh K; Figueira W F; Price P A
CORPORATE SOURCE: Department of Biology, University of California, San Diego, La Jolla, California 92093, USA.
CONTRACT NUMBER: AG07996 (NIA)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1996 Aug 9) 271 (32) 19415-20.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U49835
ENTRY MONTH: 199609
ENTRY DATE: Entered STN: 19960924
Last Updated on STN: 20010919
Entered Medline: 19960916

AB We describe the isolation of a novel protein from the conditioned medium of human articular cartilage chondrocytes in primary culture. This 39-kDa protein has the N-terminal sequence YKL, which we have termed **YKL-39**. The 1434-nucleotide sequence of the **YKL-39** cDNA predicts a 385-residue initial translation product and a 364-residue mature **YKL-39**. The amino acid sequence of **YKL-39** is most closely related to YKL-40, followed by macrophage chitotriosidase, oviductal glycoprotein, and macrophage YM-1. All five proteins share significant sequence identity with bacterial chitinases and have the probable structure of an (alphabeta)8 barrel. **YKL-39** lacks the active site glutamate, which is essential for the activity of chitinases, and as expected has no chitinase activity. The highest level of **YKL-39** mRNA expression is seen in chondrocytes, followed by synoviocytes, lung, and heart. **YKL-39** accounts for 4% of the protein in chondrocyte-conditioned medium, prostromelysin accounts for 17%, and YKL-40 accounts for 33%. In contrast to YKL-40, **YKL-39** is not a glycoprotein and does not bind to heparin.

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(FILE 'HOME' ENTERED AT 09:46:22 ON 25 JUN 2002)

FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 09:46:36 ON 25 JUN 2002
L1 0 S FTLASAETT OR HSFTLASAETTVG
L2 211 S YKL
L3 69 S L2 AND CHONDROCYTE?
L4 42 S L3 AND (AUTOIMMUN? OR RA OR ARTHRITIS)
L5 21 DUP REM L4 (21 DUPLICATES REMOVED)
L6 19 S YKL (1N) 39
L7 10 DUP REM L6 (9 DUPLICATES REMOVED)

=> end
ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
LOGOFF? (Y)/N/HOLD:y

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